CLINICAL Vs ENDOSCOPIC CORRELATION OF UPPER GASTROINTESTINAL BLEED

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CERTIFICATE

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I solemnly declare that the dissertation titled **"CLINICAL Vs ENDOSCOPIC CORRELATION OF UPPER GASTROINTESTINAL BLEED "** was done by me from March 2009 to November 2010 under the guidance and supervision of **Professor Dr.M.RAVEENDRAN M.D**

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CONTENTS

Introduction

INTRODUCTION

Gastrointestinal bleeding encompasses a broad array of clinical scenarios. The spectrum is diverse because of the multiple types of lesions that can cause bleeding, and because bleeding can occur from virtually anywhere in the gastrointestinal tract.

Additionally, gastrointestinal bleeding varies greatly in its volume and as such may be massive or trivial, and may be clinically apparent or altogether hidden.

Gastrointestinal bleeding is manifest in one or more of the following clinical scenarios: (1) bleeding is from the upper gastrointestinal tract; (2) bleeding is from the lower gastrointestinal tract; (3) bleeding is occult (ie, unknown to the patient); or (4) bleeding is clinically obvious but the site (ie, whether it is from the upper or lower gastrointestinal tract) is obscure.

Patients with occult bleeding are challenging because the patient is unaware of the bleeding and clinical clues to its cause are typically lacking. Patients with obscure bleeding are particularly challenging because their bleeding is typically recurrent and the site of bleeding is difficult accurately to identify.

Gastrointestinal bleeding results in over 300,000 hospitalizations annually. Bleeding from the upper gastrointestinal tract is approximately five times more common than from the lower gastrointestinal tract and seems to be more common in men and the elderly.

Despite a number of recent advances in the management of patients with gastrointestinal bleeding, several fundamental clinical principles remain constant, the most important of which is immediate assessment and stabilization of the patient's hemodynamic status.

Aims & Objectives

AIM OF THE STUDY

1. To find out the prevalence of nature of lesion by Upper Gastro Endoscopy in patients admitted with UGI bleed
2. To find out the prevalence of nature of lesion in patients with mild, moderate and major bleed
3. To identify the risk factors associated with poor outcome
4. To assess the nature of lesions in patients with UGI bleed presenting with co-morbidities
5. To find out the nature of lesions in patients with UGI bleed and correlate with their clinical presentation

Review of Literature

REVIEW OF LITERATURE

Gastrointestinal bleeding is one of the common gastroenterological emergencies. The spectrum is diverse because of the multiple types of lesions that can cause bleeding, and because bleeding can occur from virtually anywhere in the gastrointestinal tract[1]. Bleeding from the upper gastrointestinal tract is approximately five times more common than from the lower gastrointestinal tract and seems to be more common in men and the elderly[2,3]. Despite a number of recent advances in the management of patients with gastrointestinal bleeding, several fundamental clinical principles remain constant, the most important of which is immediate assessment and stabilization of the patient's hemodynamic status. Thereafter, a careful history and physical examination follows, and with it the etiology and source of bleeding predicted. Specific investigation then follows and further delineates the source of bleeding.

Once the bleeding site is identified, it must be stopped and treated. Subsequently, recurrent bleeding should be prevented.

CLINICAL PRESENTATION

The clinical presentation of patients with gastrointestinal bleeding typically reflects the site, etiology, and rate of bleeding. Gastrointestinal tract bleeding may manifest in one or more ways.

Hematemesis and melena are the most common manifestations of upper gastrointestinal bleeding. Hematemesis is defined as vomiting of blood and is caused by upper gastrointestinal bleeding from the esophagus, stomach, or proximal small bowel. Blood may be bright red or it may be old and take on the appearance of coffee grounds. Melena is defined as passage of black, tarry, and foul-smelling stools. The black, tarry character of melena is caused by degradation of blood in the more proximal colon.

INITIAL PATIENT ASSESSMENT

When a patient is found to have one of the previously mentioned manifestations of gastrointestinal bleeding, the first step in management should be to assess the severity of bleeding. Assessment of the patient's hemodynamics should be emphasized. Ongoing assessment of the vital signs further focuses resuscitation efforts, and also provides important prognostic information.

Table 1:

Hemodynamics, vital signs and blood loss

Hemodynamics vital sign	% Blood loss (fraction of intravascular volume)	Bleed type
Shock (resting hypotension)	20-25	Massive
Postural (orthostoatic tachycardia or hypotension)	10-20	Moderate
Normal	< 10	Minor

RESUSCITATION[4]

The more severe the bleeding, the more vigorous the resuscitation efforts should be. In patients who have any evidence of haemodynamic instability, two large - bore intravenous catheters should be placed immediately. Crystalloid (normal saline or lactated Ringer's solution) should be infused as rapidly as the patient's cardiovascular system allows to restore the vital signs toward normal. ICU monitoring is indicated in hemodynamically unstable patients. Supplemental oxygen by nasal cannula or facemask should be given liberally. Vital signs and urine output should be monitored closely, and in selected situations (for patients with underlying cardiopulmonary disease) central venous monitoring is helpful. The importance of aggressive ICU monitoring and resuscitation

has been emphasized by investigation suggesting that it may decrease mortality[1].

In addition, patients must typically undergo blood transfusion as needed. If the patient has subnormal tissue oxygenation, transfusion should be aggressive. Patients with continued instability in vital signs, continued bleeding, symptoms of poor tissue oxygenation, or persistently low hematocrit values (20%–25%) likewise should probably be transfused continuously. It is most appropriate to raise the hematocrit to a level of 30% in elderly patients, whereas in younger, otherwise healthy patients, hematocrit values in the 20% to 25% range may be satisfactory; in those with portal hypertension, it should not be above 27% to 28%. Transfusion should be with packed red blood cells, except in rare circumstances where whole blood transfusions may be used. Serial hematocrits are not a substitute, for ongoing clinical assessment of the hemodynamics.

HISTORY, SYMPTOMS, AND SIGNS

Historical features important in assessing the etiology of gastrointestinal bleeding are shown below

- Age
- Prior bleeding
- Previous gastrointestinal disease
- Previous surgery
- Underlying medical disorder (especially liver disease)
- Nonsteroidal anti-inflammatory drugs (NSAIDs), aspirin (ASA)
- Abdominal pain
- Change in bowel habits
- Weight loss or anorexia
- History of oropharyngeal disease

Symptoms

Hematamesis and Melena

Postural giddiness

Palpitation

Sweating

Signs

BP: Resting hypotension and postural hypotension and tachycardia

Signs of liver disease: Jaundice, spider angiomata, palmar erythema, dupuytren's contracture.

Acanthosis nigricans (underlying malignancy)

Abdominal mass and tenderness

LABORATORY EVALUATION[5]

During initial evaluation the following tests are of urgent importance.

Hemoglobin

Hematocrit

Blood urea nitrogen[5]

Serum creatinine[5]

CLINICAL LOCALIZATION OF BLEEDING

The localization of bleeding should begin with the history and physical examination and should be focused immediately, during hemodynamic stabilization. Hematemesis denotes an upper gastrointestinal source of bleeding. Melena indicates that blood has been in the gastrointestinal tract for extended periods of time and is usually the result of upper gastrointestinal bleeding, but its source may be the distal small bowel or even the ascending colon. In the latter instance, the volume of bleeding is too little to cause hematochezia[6] but sufficiently

large to provide hemoglobin for degradation. The nasogastric lavage has been commonly used to help differentiate upper from lower gastrointestinal bleeding[7.8] A bloody aspirate indicates that the upper gastrointestinal tract is the source of bleeding, because the false-positive rate is extremely low, and is usually caused by nasogastric trauma[8].

Assessment of vital signs and the use of bedside diagnostic criteria as previously mentioned is a more effective means to determine the activity of bleeding. If there is any question about the location of bleeding in a patient with hematochezia[6], especially in patients with hemodynamic instability, a nasogastric tube should be placed. Other clues that can help localize an upper gastrointestinal source of bleeding include hyperactive bowel sounds and an elevation in the blood urea nitrogen level out of proportion to creatinine.

DIAGNOSIS AND THERAPY

Diagnostic tests play a central role in the evaluation of patients with gastrointestinal bleeding. The major categories of tests available include the following: (1) endoscopy; (2) barium radiographs; (3) radionuclide imaging; (4) angiography; and (5) miscellaneous tests (ie, abdominal CT scanning). The radiographic tests allow diagnosis only, whereas endoscopic tests allow both diagnosis and therapy. The importance of endoscopic therapy is emphasized by studies performed

before the advent of endoscopic therapy, which demonstrated that endoscopy per se did not affect outcome for patients with upper gastrointestinal bleeding[9].

A major goal of treatment is to stop active bleeding and prevent recurrent bleeding. The major forms of therapy include (1) pharmacologic, (2) endoscopic, (3) angiographic, and (4) surgical. The use of each of these modalities has undergone tremendous change over the past two decades. Traditionally, UGIB is categorized as being either variceal or non variceal for planning appropriate therapeutic strategy.

NON VARICEAL BLEED

EPIDEMIOLOGY

Several population-based and prospective studies support peptic ulcer disease (PUD) being the most common cause of acute UGIB[10]. PUD traditionally refers to either gastric or duodenal ulcers, but under a broad heading of ‘‘ulcers’’ some investigators also include esophageal ulcers[11] Approximately 50% of all cases of acute UGIB are attributed to PUD, and it has long been suggested to be the most common cause of nonvariceal UGIB[12,13,14]. Recent evidence suggests, however, that the incidence of PUD as a cause of acute UGIB may either be decreasing or is underreported. Widespread proton pump inhibitor prescribing and

Helicobacter pylori eradication protocols also likely contribute to this observed downward incidence trend of PUD causing nonvariceal UGIB.

Classically, Mallory-Weiss tears are mucosal lacerations at the gastroesophageal junction or in the cardia of the stomach[15]. These lesions can be associated with repeated retching or vomiting and are another important cause of nonvariceal UGIB. It is estimated that 5% to 15% of all cases of acute UGIB are secondary to Mallory-Weiss tears[16,17,18]. Most bleeding episodes caused by Mallory-Weiss tears are self-limited and do not require endoscopic hemostasis .Vascular ectasias, also referred to as ‘‘angiomas,’’ ‘‘arteriovenous malformations,’’and ‘‘angiodysplasia,’’ are another source of acute and chronic nonvariceal UGIB[19]. Vascular ectasias are the underlying etiology of acute UGIB in approximately 5% to 10% of cases and the severity of bleeding can also range from trivial to severe.

Isolated vascular ectasias are endoscopically different than the diffuse linear array seen in gastric antral vascular ectasia, also referred to as ‘‘watermelon stomach’’[20,21]. Gastric antral vascular ectasia(GAVE) is thought to be a distinct clinical entity from portal hypertensive gastropathy and is characterized by red stripes interposed by normal-appearing mucosa generally noted in the gastric antrum[22].

Dieulafoy's lesion is a rare etiology in acute UGIB. Their histopathologic description is a ''caliber-persistent artery'' in the submucosal tissue[23]. These lesions represent the etiology for nonvariceal UGIB in less than 5% of all UGIB cases[24,25]. On endoscopy, a Dieulafoy's lesion is akin to a visible vessel protruding from an ulcer, yet without an underlying ulcer. Neoplasms, both malignant and benign, are another infrequent cause of nonvariceal UGIB and comprise less than 5% of all UGIB cases[26]. Although neoplasms make up a small fraction of overall bleeding episodes, UGIB can be a common presenting sign for a neoplasm and should be part of the differential diagnosis[27]. The lesion can be a primary malignancy, such as esophageal, gastric, or duodenal adenocarcinoma; esophageal squamous cellcarcinoma; gastric or duodenal lymphoma; or a gastrointestinal stromal cell tumor.

Aortoenteric fistula (aortic aneurysm repair)[28], hemobilia[29] and haemosuccus pancreaticus[30] are other rare causes of nonvariceal UGIB which should also be considered in any differential diagnosis. Finally, iatrogenic injuries secondary to endoscopic procedures, such as percutaneous endoscopic gastrostomy tube placement, are also rare causes of nonvariceal UGIB[31]. 10%, however, and these figures have not dramatically changed.

DIAGNOSIS

Clinical Presentation and Patient Triage

A thorough medical history and careful physical examination are critical in the assessment of an individual presenting with gastrointestinal bleeding. Details from the patient history can help mold the differential diagnosis and stratify patient outcomes. A history of taking nonsteroidal anti-inflammatory drugs, aspirin, antiplatelet agents, or anticoagulation therapies, such as warfarin, are important pieces of information to acquire during the history. A history of documented PUD with previous UGIB, known H.pylori infection and compliance with proton pump inhibitor therapy are also important issues to address. Furthermore, the aforementioned relationship between advanced age, chronic renal insufficiency, valvular heart disease and vascular ectasias should be noted. Advanced patient age also increases the likelihood of a gastrointestinal neoplasm as a possible etiology[32,33].

Nasogastric tube aspiration can be an important component of the evaluation of UGIB. Studies evaluating the usefulness of nasogastric tube aspiration in predicting high-risk lesions, such as a peptic ulcer with active bleeding or a visible vessel, have produced large variations in sensitivity, specificity, and positive and negative predictive values. In general, overtly bloody nasogastric tube aspirates are correlated with

high-risk endoscopic lesions in the upper gastrointestinal tract [34,35,36] Clinicians often question the need for an nasogastric tube in the setting of melena or hematemesis. Nasogastric tube placement does not serve a purely diagnostic purpose, however, and can be useful for gastric lavage and has the potential to minimize the aspiration risk from a blood-filled stomach in preparation for endoscopy[37]. Specifically, gastric lavage has been shown to improve visualization of the gastric fundus when performed before endoscopy for acute UGIB[38]. To risk stratify individuals presenting with acute UGIB better, risk scoring tools have been developed to facilitate patient triage, predict risk of rebleeding and mortality, evaluate need for ICU admission, and determine need for urgent endoscopy[39]. These scoring tools have been almost exclusively used in research studies, and are uncommonly applied in everyday clinical practice[40]. Some risk scoring tools, such as the Blatchford Score, use laboratory findings, patient vital signs at presentation, and other clinical variables without the use of endoscopy[41]. The complete Rockall Score identified significantly more low-risk patients with acute UGIB than either the clinical Rockall Score or the Blatchford Score. Risk stratification may be important because of the potential to minimize the unnecessary use of hospital-based services, iatrogenic complications, and worker absenteeism.

Gastrointestinal Endoscopy

Endoscopy is the best tool for both the diagnosis and ultimately as a therapeutic measure for patients with acute UGIB. Improvements in endoscopic technology and operator skill have clearly reduced the need for surgery and interventional radiology as diagnostic and therapeutic procedures for UGIB. Patient positioning before and during the procedure can assist with improving visibility during endoscopy[42]. Positioning the patient with the bleeding point in the most superior position can help clear the endoscopic field by allowing blood to flow away from the point of bleeding. Reverse Trendelenburg positioning and rolling the patient from the left lateral decubitus position to the right and back can also be used to move clots and blood away from dependent areas in the stomach. The choice of endoscope is also a critical aspect of making an accurate diagnosis for nonvariceal UGIB. A large single-channel or double channel therapeutic endoscope should be used in all cases of suspected acute UGIB. This technique should be used both for the ability to suction larger volumes of gastroduodenal contents and for the potential to provide hemostasis therapy using a large-size (10F catheter) thermal probe or mechanical clipping device.

Early endoscopy, generally defined as within 24 hours of hospitalization has been shown to reduce resource use, decrease

transfusion requirements, and shorten hospital stay[43,44]. There is conflicting evidence whether or not performing endoscopy even earlier (eg, in the emergency department) can further decrease resource use and minimize health care costs. Lee and colleagues[45] demonstrated a significant decrease in hospital costs and duration of stay with endoscopic triage performed in the emergency. The presence of blood in the stomach on upper endoscopy is an important finding when assessing risk initially.

Table 2:

Prevalence and Outcomes of PUD using endoscopic stigmata[46]

Endoscopic Characteristics	Forrest Classifications	Prevalence % (range)	Rebleeding % (range)	Surgery % (range)	Mortality % (range)
Clean base	III	42(19-52)	5(0-10)	0.5 (0-3)	2(0-3)
Pigmented flat spot	IIC	20(0-42)	10(0-13)	6(0-10)	3(0-10)
Adherent clot	IIB	17(0-49)	22(14-36)	10(5-12)	7(0-10)
NBVV	IIA	17(4-35)	43(0-81)	34(0-56)	11(0-21)
Active bleed	IA	18(4-26)	55(17-100)	35(20-69)	11(0-23)

Active bleeding (spurting or oozing) or nonbleeding visible vessels seen on endoscopy have the highest potential for rebleeding with rates of

approximately 55% and 43%, respectively. This knowledge can guide the decision for discharge, hospital admission, or admission to an ICU. Despite the importance of making an accurate diagnosis, evidence suggests that there is frequent intraobserver variability on grading endoscopic stigmata[47,48]. If there is clinical evidence of recurrent bleeding after primary hemostasis has been achieved, esophagogastroduodenoscopy should be repeated with a view toward repeat hemostasis if needed[49]. In UGIB secondary to PUD, rebleeding occurs in approximately 10% to 30% of patients who have high-risk stigmata (active bleeding, nonbleeding visible vessel, adherent clot) at the time of initial esophagogastroduodenoscopy and who receive endoscopic hemostasis[50]. In addition, evidence from recent studies suggests that performing a second-look endoscopy (regardless of any clinical evidence of recurrent hemorrhage) in patients with high-risk ulcer stigmata at the time of their initial esophagogastroduodenoscopy may decrease rebleeding rates, need for surgery, and health care costs[51,52]. The feasibility and importance of this in actual clinical practice, however, may be dependent on the health care setting of the patient and provider.

Using ultrasound as an accessory modality for cases of UGIB is not a new Concept[53]. Much of the literature surrounding the use of endoscopic ultrasound for UGIB involves gastroesophageal varices and

other clinical issues related to portal hypertension[54,55,56]. The literature on endoscopic ultrasound in nonvariceal UGIB is growing, however, and reports exist of this technique being used as an adjunctive diagnostic tool in a variety of settings including peptic ulcer hemorrhage, Dieulafoy's lesion, and evaluating hemobilia[57,58]. A Doppler ultrasound probe can be passed through the accessory channel of a therapeutic endoscope. A persistent Doppler ultrasound signal after endoscopic hemostasis in PUD has been associated with a higher rebleeding rate, and some authors endorse evaluating for the presence of a Doppler signal before and after hemostasis therapy in PUD so as to ensure better complete hemostasis[59].

Additional Modalities

Angiography and technetium scanning are useful alternatives when endoscopy has not yielded a definitive diagnosis or is unable to be performed. Other radiographic studies may also be useful when making the diagnosis of UGIB, but not usually in the acute setting. An esophagram, or upper gastrointestinal series, and small bowel follow-through were used more for the diagnosis of UGIB before the advances in endoscopic technologies over the past two decades and generally are no longer used as part of the diagnostic evaluation of persons with acute UGIB[60,61,62].

Wireless capsule endoscopy is now being used as a diagnostic tool for a variety of gastrointestinal disorders including Crohn's disease, celiac disease, and obscure gastrointestinal bleeding[63,64]. At the present time, however, it is not a practical modality to use in acute UGIB.

TREATMENT

Nonvariceal upper gastrointestinal bleeding (UGIB) remains a common emergency for gastroenterologists with an annual incidence of 50 to 150 per 100,000 of the population. Mortality from UGIB is around 10%, and may reach 35% in patients hospitalized with another medical condition. Serious comorbidity remains an independent risk factor for UGIB mortality, which is often attributable to increasing age and associated illnesses. A recent time trend analysis by a Dutch group has demonstrated a decrease in incidence of UGIB (from 61.7 per 100,000 per year in 1993–1994 to 47 per 100,000 per year in 2000), but has not demonstrated a reduction in mortality or rebleeding rates[65], even though there have been significant advances in medical and endoscopic management of serious UGIB. An ageing population with potentially serious comorbidities helps to explain the lack of concordance between the overall population incidence and mortality rate for UGIB. Patients over 80 years of age now account for around 25% of all UGIB and 33% of UGIB occurring in hospitalized patients.

ETIOLOGY OF NONVARICEAL UPPER GASTROINTESTINAL BLEEDING

The causes and historically quoted frequencies of nonvariceal UGIB are shown in Table 3.

Table 3:

Causes of nonvariceal upper gastrointestinal bleeding

Diagnosis	Incidence (%)
Peptic ulcer	30-50
Mallory-Weiss tear	15-20
Erosive gastritis or duodenitis	10-15
Esophagitis	5-10
Malignancy	1-2
Angiodysplasia or vascular malformations	5
Other	5

There is controversy regarding the relative contribution of peptic ulcer bleeding to overall UGIB rates. Recent data from the Clinical Outcome Research Initiative suggest that the frequency of peptic ulcer as a cause of UGIB may have been overestimated. In 7822 endoscopies performed for UGIB, peptic ulcer was the likely cause in only 1610 patients (20.6%). Data from the Canadian Registry on Nonvariceal Upper Gastrointestinal Bleeding and Endoscopy, however, identified peptic ulcers in 50% of patients presenting to community and tertiary care

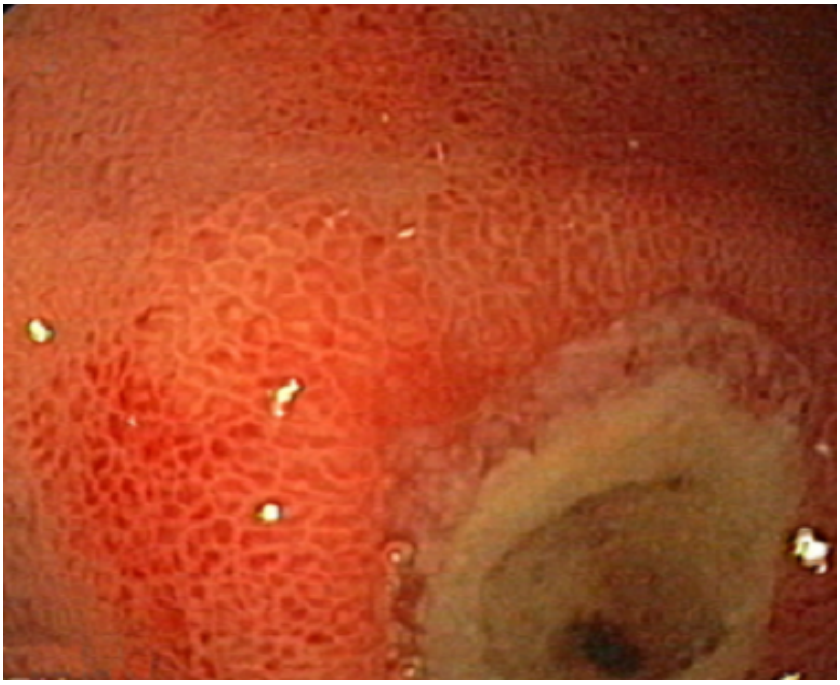


Fig 1: Duodenal Ulcer

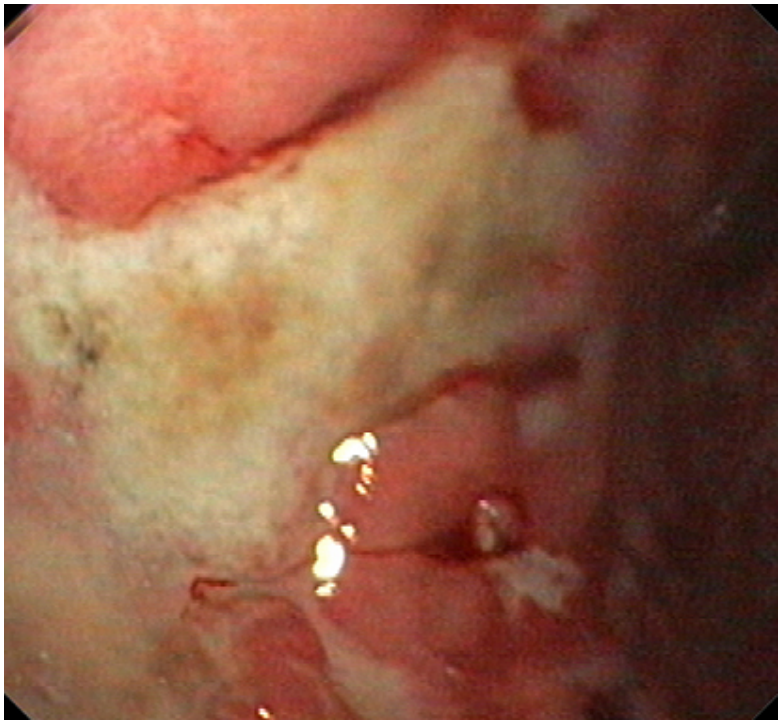


Fig 2: Gastric ulcer

institutions between 1999 and 2002. Regardless of the historical frequency of peptic ulcer bleeding, the incidence of peptic ulcer disease should decline with more widespread Helicobacter pylori eradication. In addition, widespread use of cyclooxygenase-2–specific nonsteroidal anti-inflammatory drugs may also affect peptic ulcer risk, although prescription of this particular class of drugs worldwide has been severely affected by recent statements by the US Food and Drug Administration and other national drug monitoring organizations.

CLINICAL RISK ASSESSMENT

One of the major challenges of managing UGIB involves identifying patients who are at high risk of rebleeding and death; conversely, identifying patients who are suitable for early discharge and outpatient endoscopy is also important for effective resource use. Several clinical scoring systems have been developed to help predict outcome for patients with a view to improving patient management and promoting cost-effective use of resources. In most published scoring systems, a combination of clinical, laboratory, and endoscopic variables are weighted to produce a score that predicts the risk of mortality, recurrent hemorrhage, need for clinical intervention, or suitability for early discharge. The most commonly used systems (Rockall score, the Baylor

bleeding score, Blatchford score) were recently excellently reviewed by Das and Wong[66] . Several factors are associated with poor outcome from UGIB and may be related to the patient's presentation and comorbidities, or to the behavior of the ulcer:

Shock

Melena

Anemia at presentation

Significant fresh blood in vomit, gastric aspirate, or rectum

Concurrent sepsis

General poor health

Liver, renal, cardiac disease

Large ulcer size

Persistent bleeding despite endoscopic therapy

Recurrent bleeding

Inclusion of endoscopic stigmata of recent hemorrhage (SRH) that relate to increased risk of rebleeding and death into scoring systems increases the sensitivity for predicting patients at high or low risk of adverse events compared with nonendoscopic assessments[67,68]. In addition, early endoscopy based triage may allow safe and early discharge of low-risk patients with no increased rate of rebleeding or mortality. Risk stratification using nonendoscopic parameters has the advantage that it

can be performed readily on initial presentation in the emergency department, however, and if early endoscopy, which requires skilled staff and resources, is not available, appropriate initial risk assessment can be made. Clearly, more studies are required to clarify the role of endoscopy in early risk assessment. More generally, care must be taken when applying a risk stratification scoring system to any patient population not represented by the original studies, because racial, cultural, or ethnic factors may affect a populations' risk[69].

INITIAL MANAGEMENT

Resuscitation and optimization of comorbid conditions are vital in the initial management of patients before endoscopy. Transfusion of blood and blood products may be necessary and patients often require management in an intensive care setting. Endotracheal intubation remains controversial in significant nonvariceal UGIB. The endoscopist's task is made easier and the risk of massive aspiration in a patient with reduced level of consciousness is reduced if a patient is intubated; however, evidence of a reduction in acquired pneumonia or cardiopulmonary events is lacking . The presence of blood-stained nasogastric aspirate can be used to predict the presence of high-risk lesions and nasogastric tube insertion should be considered for some patients. The optimum timing of

endoscopy remains a balance between clinical need and resources, but endoscopy performed within 24 hours of hospital admission has been shown to reduce the length of hospital stay and may reduce the likelihood of rebleeding or surgical intervention in the highest risk patients[70].

ENDOSCOPIC ASSESSMENT

Endoscopic SRH associated with a higher risk of rebleeding, surgical intervention, and death have been well defined[71]. High-risk lesions, such as actively bleeding ulcers, nonbleeding visible vessels (NBVV), and adherent clots[72] require aggressive intervention because ulcer rebleeding is associated with a 5- to 16-fold increase in mortality , and effective endoscopic management can substantially reduce this risk. The rebleeding rate of ulcers with a clean base or red or blue spots is low, and endoscopic intervention is usually not recommended[69]. Although actively bleeding vessels are consistently identified by endoscopists, this is not the case for other SRH, particularly NBVV and flat pigmented spots. Attention has turned to alternative approaches to assess lesions more objectively. Doppler examination of ulcers was assessed as a means of obtaining objective evidence of rebleeding risk in 100 patients admitted with UGIB but not bleeding at the time of index endoscopy. Doppler findings were compared with the Forrest classification of the ulcer[73]. Ulcers were assessed for the presence of blood vessels and were

considered to be Doppler-positive if a vessel no deeper than 1 mm was identified. Doppler-positive ulcers and those in the Forrest group with adherent clot or visible vessels were treated endoscopically. There was agreement between the Forrest classification and Doppler in only 58% of cases. Rebleeding, requirement for surgery, and mortality rate were all significantly lower in the Doppler-assessed group. The authors suggest that Doppler assessment can guide appropriate endoscopic intervention for patients with NBVV. Technical and resource limitations, however, mean this technique is unlikely to be widely available for some time. Not infrequently, excessive blood in the upper gastrointestinal tract may preclude an accurate endoscopic diagnosis. A retrospective study by Cheng and coworkers identified 25 of (1.7%) 1459 patients where a diagnosis could not be made endoscopically because of blood obscuring the examination field. Not surprisingly, these patients had a significantly higher rate of complications, rebleeding, need for surgery, and mortality. The authors stress the importance of good preparation along with the removal of blood during the procedure. Bolus administration of intravenous erythromycin before endoscopy has been shown to clear the stomach of blood, thereby increasing the likelihood of successful hemostasis and reducing the need for further interventions[74,75]

ENDOSCOPIC MANAGEMENT

Endoscopic intervention is beneficial in high-risk patients with UGIB, reducing the rate of rebleeding, need for surgical intervention, and mortality. It is likely that most hemostatic techniques are equally effective when used alone. Recent research has focused on the role of combination therapies and newer mechanical means of homeostasis[76]. Most of the following text refers to peptic ulcer bleeding but may be applicable in other causes of nonvariceal UGIB.

Injection Therapy

Injection of dilute (1:10,000) adrenaline in 1-mL aliquots around the bleeding points results in hemostasis in up to 100% of patients with bleeding peptic ulcers, probably by a combination of vascular tamponade and vasoconstriction, with a concomitant reduction in rebleeding rates from 40% to 15%[77,78]. The dose of adrenaline required to achieve hemostasis is probably dependent on the individual patient; however, in a study of 156 patients with Forrest type I or IIa lesions a larger volume (13–20 versus 5–10 ml) resulted in less rebleeding (15.4% versus 30.8%) Although injection with adrenaline is successful in achieving initial hemostasis, the published rebleed rates of 15% to 36% remains relatively high. Attention has focused on alternative techniques (eg, heat or

mechanical) or combination therapy to determine if there is any additional benefit. Sclerosants, such as ethanol, polidocanol, and ethanolamine, have been used to promote vessel thrombosis, but evidence to date suggests these agents are no better, and may have more risk, than adrenaline. In one study, ethanol injection alone was shown to have a rebleeding rate as low as 4% however, most other published studies have demonstrated similar or worse rates of hemostasis than adrenaline alone. A combination of adrenaline and ethanol may improve hemostasis and shorten duration of hospital stay for patients with spurting hemorrhage[79].

Thrombin-fibrinogen mixture (fibrin-sealant glue) does not seem to confer any additional benefit beyond adrenaline alone when used in a one-off basis in combination with adrenaline injection for patients with high-risk peptic ulcers, although it may be of particular use in patients with active bleeding. A study involving 51 patients with active bleeding or NBVV demonstrated lower rebleeding rates with a single treatment with combination adrenaline and fibrin sealant compared with adrenaline alone, although there was no difference in mortality, transfusion requirements, surgery, or duration of hospital stay. Repeated injection of fibrin glue following treatment with dilute adrenaline in patients with active bleeding or NBVV was subsequently compared with single application of fibrin glue or polidocanol following adrenaline injection.

Patients underwent daily endoscopy until the ulcer base was clean or covered in hematin. Patients in the repeat treatment group had significantly higher rates of hemostasis with less rebleeding compared with the polidocanol group, although mortality rates were not reduced. The major drawback of this schedule is the cost incurred by repeated daily procedures.

In another study, endoscopic intervention with a combination of adrenaline injection and 600 to 1000 IU human thrombin has been shown to be more effective than injection of adrenaline alone, with a reduction in rebleeding (4.5% versus 20%), transfusion requirement, and mortality[80]. Injection with N-butyl-2-cyanoacrylate has been shown to be effective for control of variceal bleeding, but its role in nonvariceal UGIB remains uncertain. In a small study of 32 cases it was no more effective than injection with dilute adrenaline for control of bleeding ulcers. More recently, Lee and coworkers showed significantly lower rebleeding rate for patients with Forrest type Ia lesions treated with N-butyl-2-cyanoacrylate compared with injection with hypertonic saline-adrenaline injection. There was no overall benefit in the use of N-butyl-2-cyanoacrylate with regards to hemostasis rates, emergency surgery, or mortality. Arterial embolization is a recognized complication of this treatment, and occurred in 2 of 63 patients in the treatment group. The

authors recommend N-butyl-2-cyanoacrylate injection only as a measure of last resort before surgery because of potentially fatal adverse effects. Thermal Techniques Several thermal techniques have been used for the control of nonvariceal UGIB. Homeostasis is achieved by compression of the artery during heating (coaption) and the effect of heat on tissue.

Noncontact thermal techniques

Laser (neodymium:yttrium-aluminum-garnet) and argon plasma coagulation are the only noncontact thermal therapies currently available. Argon plasma coagulation causes hemostasis by conducting a high-frequency electricalcurrent through a beam of ionized argon gas, resulting in superficial tissue damage and coagulation. Although the technique is generally safe and relatively straightforward, the efficacy has yet to be fully determined. A prospective observational study into the use of argon plasma coagulation in 254 patients with nonvariceal UGIB revealed initial hemostasis rates of 75.9% and rebleeding rates of 5.7% with argon plasma coagulation alone. When a second endoscopic technique was added, initial hemostasis was achieved in 99.6%. In the only comparative randomized trial involving argon plasma coagulation in nonvariceal UGIB, rates of hemostasis, rebleeding, emergency surgery, and 30-day mortality were comparable with the heater probe, although the numbers in

this study (N ¼ 41) were too small to detect a difference . Chau and coworkers compared combination treatment with adrenaline and heater probe with adrenaline and argon plasma coagulation in a prospective, randomized, and controlled trial involving 185 patients with bleeding peptic ulcers. There was no significant difference in primary hemostasis, procedure duration, rebleeding, requirement for surgery, 30-day mortality, or ulcer healing at 8 weeks, suggesting that combination therapy with adrenaline and argon plasma coagulation is as effective as heater probe in high-risk patients with bleeding ulcers. Laser therapy has been shown to be as effective as injection with epinephrine- polidocanol , but because of technical constraints of the technique, laser therapy is not routinely used in the management of nonvariceal UGIB.

Contact thermal techniques

Bipolar electrocoagulation and heater probe thermocoagulation use thermal contact to achieve hemostasis by compression of the vessel and coaption. A bipolar electrocoagulation device may include an injector-irrigator component (eg, Gold probe, Boston Scientific, Boston, MA) to allow injection of adrenaline and irrigation of the culprit lesion. Bipolar electrocoagulation has been shown to reduce the rebleeding rate when compared with normal saline injection in high-risk bleeding ulcers, and in

combination with adrenaline in type IIb ulcers. Combination therapy with heater probe thermocoagulation and adrenaline in the treatment of actively bleeding peptic ulcers resulted in hemostasis in up to 98.6%, with rebleeding in 8.2%. In another study, however, there was no significant difference in rates of rebleeding, requirement for surgery, and length of hospital stay when compared with adrenaline alone. Subgroup analysis, however, did illustrate benefit in patients with Forrest Ia lesions, with dual therapy resulting in significantly lower rebleeding rates and non significant reductions in emergency surgery and length of hospital stay. When used alone, heater probe thermocoagulation was not superior to combination treatment with adrenaline and polidocanol in patients with Forrest type I, IIa, and IIb ulcers. Heater probe thermocoagulation (HPC) in combination with thrombin was compared to HPC and placebo in 247 patients with bleeding peptic ulcers and was found to confer no benefit when compared with the placebo arm with regards to hemostasis, rebleeding rates, requirement for surgery, adverse events, or mortality Mechanical Techniques Mechanical methods of achieving hemostasis are often used in variceal UGIB.

Endoloops and particularly clips (eg, the Hemoclip [Teleflex Medical, Research Triangle Park, NC]), however, are likely to play an

increasing role in the control of nonvariceal UGIB. Hemostasis using endoclips involves deployment of a clip to achieve vascular compression. So far the Hemoclip has been safe and effective, achieving homeostasis rates of up to 100%. Comparative studies with other endoscopic techniques suggest lower rebleeding rates than adrenaline injection, ethanol, or hypertonic saline-epinephrine. The additional benefit of adrenaline with a mechanical method is unclear. A randomized comparative study of injection of epinephrine-polidocanol and Hemoclip versus Hemoclip alone, however, showed clipping to be inferior to combination Hemoclip– adrenaline injection therapy in the treatment of bleeding peptic ulcers[81]. Chung and coworkers found the Hemoclip to be an effective method for hemostasis and safer than hypertonic saline-epinephrine, and combination treatment with injection therapy and Hemoclips was equivalent to either treatment alone for control of bleeding. Rebleeding rates and the need for surgery were higher, however, in the adrenaline group. A potential limitation of the Hemoclip is the technical difficulty in applying the clips to difficult-to-reach lesions, particularly those high on the gastric lesser curve or posterior wall of the duodenum. This was demonstrated in a comparative study of Hemoclip with heater probe thermocoagulation in which the overall rates of hemostasis were 85% and 100%, respectively. In the subgroup of

difficult-to-approach lesions the homeostasis rate fell to 30% and 82%, respectively. Rotatable and more versatile endoclips may help to lessen this problem. In addition, devices that can deploy multiple or stronger clips are needed. Two small studies have evaluated the role of Hemoclips for control of bleeding caused by Dieulafoy's lesion. Hemostasis was generally successful and there was a trend toward reduction in the need for repeat procedures.

Endoscopic band ligation is currently technically easier to use than endoclips and has been shown to be safe and effective for control of small lesions in 19 patients with acute peptic ulcer bleeding. Rubber band ligation has recently been assessed in a small group of patients with UGIB secondary to Dieulafoy's lesion and found to be as effective as injection with or without thermal therapy.

Adherent Clots

Special mention needs to be made regarding the problem of adherent clots. A subgroup analysis of patients with adherent clots in early endoscopic studies demonstrated little or no benefit of endoscopic therapy for ulcers with adherent clots. A subsequent meta-analysis showed significant benefit only in patients with active bleeding or NBVV. A randomized controlled trial to assess endoscopic intervention in

patients with severe UGIB and adherent clot randomized 32 patients to medical or combination therapy following irrigation of the clot. Endoscopic therapy consisted of adrenaline injection shaving of the clot with cold guillotine, and bipolar coagulation of the underlying stigmata. Combination therapy was shown to be safe with significantly less early rebleeding compared with medical therapy, although the small sample size, unexpectedly low rebleed rates in the treatment group (0%), and unequal distribution of confounding factors in the two groups means that caution needs to be taken when extrapolating the results. In addition, various studies have shown intraobserver variation in the labeling of SRH, and the degree of clot adherence may vary depending on the extent of clot irrigation. For instance, in one study 5 minutes of irrigation by a bipolar probe was found to remove clot in 43% of patients, whereas irrigation with a syringe only removed 9% of the clot. In another study, 10 seconds of irrigation with WaterPik (Teledyne, Fort Collins, CO) removed clots in a further 26% of patients. Placement of a newly designed transparent irrigating hood that allows forceful irrigation yet maintains a reasonable endoscopic view may prove useful for clot removal. Although the optimum technique for clot removal is unclear, the value of clot removal is clear, because high-risk SRH may be exposed in the underlying ulcer in a further 30% of patients. Rebleeding rates for

untreated ulcers with adherent clot are reported as 20% and in the group where clots remained was 8%, which is similar to that expected in low-risk lesions, such as flat pigmented spots. Current practice among experienced endoscopists involves targeted irrigation of an adherent clot to dislodge it, if possible, followed by treatment of the underlying lesion.

SECOND-LOOK ENDOSCOPY AND ENDOSCOPIC RETREATMENT

Several studies investigating the role of routine second-look endoscopy following endoscopic treatment have shown no benefit with regards to clinically significant outcomes for unselected patient populations, although there may be a role in high-risk patients. Repeat endoscopy is indicated if there is clinical evidence of rebleeding or if the initial procedure was unsuccessful or partially successful, although this depends on local endoscopic and surgical expertise[69,82]. In expert centers, endoscopic retreatment is associated with fewer complications, less need for surgery, and no increased mortality risk compared with surgery.

FUTURE DIRECTIONS IN ENDOSCOPY

Endoscopic suturing a variety of endoscopic suturing devices have been developed primarily for gastroplication in patients with gastroesophageal reflux. Endoscopic suturing for UGIB is an attractive

prospect, but further development of new devices is required before suturing for UGIB can be widely adopted. Such issues as the device size and maneuverability, and precise control of suture depth, need to be addressed.

Cryotherapy

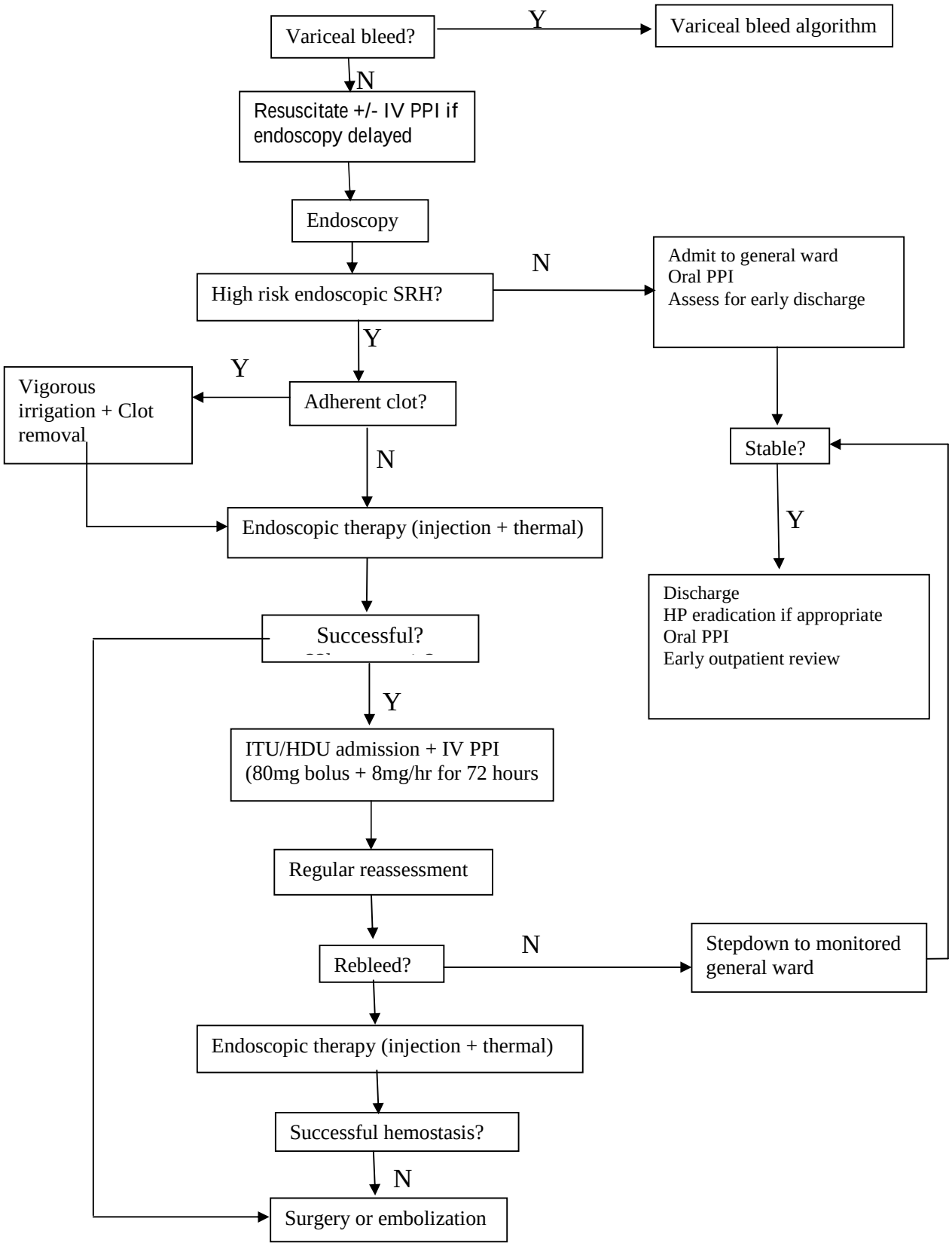
Cryosurgery involves freezing tissue to achieve a therapeutic response. Gastric freezing to achieve hemostasis during variceal and nonvariceal bleeding has been possible for several decades, although evidence of therapeutic benefit from the original techniques was lacking. More recently, delivery systems for liquid nitrogen or nitrous oxide have made endoscopic cryotherapy possible for bleeding and other applications. Delivery of nitrous oxide to result in cryotherapy relies on the Joule-Thompson effect: rapid expansion of compressed gas results in a drop in temperature of the gas. This allows noncontact therapy to localized or diffuse vascular lesions. The technique remains experimental, but it seem to be safe and effective for radiation proctitis and vascular malformations, and there may be potential use in other gastrointestinal vascular lesions.

PROTON PUMP INHIBITORS

In vitro studies of the effect of gastric pH on platelet aggregation and coagulation provide the rationale for acid suppression in UGIB. If gastric pH is maintained above pH6 (by infusional proton pump inhibitors), platelet aggregation is optimized and fibrinolysis relatively inhibited, thereby potentially improving the likelihood of clot stability at an ulcer site. Individual trials of H2 receptor antagonists have generally failed to demonstrate a clinical benefit in UGIB , although a meta-analysis has suggested a weak effect . Several studies have evaluated intravenous proton pump inhibitors for nonvariceal UGIB; unfortunately, these trials are heterogeneous in terms of patient population, regimen of proton pump inhibitor, and timing or type of endoscopic intervention, making comparisons difficult. Five meta-analyses of proton pump inhibitors in nonvariceal UGIB have now shown a benefit, however, in terms of rebleeding and need for surgery, but not for mortality[83-87]. The usual intravenous regime for omeprazole therapy in the more robust studies was an 80-mg intravenous bolus of omeprazole followed by a continuous infusion of 8 mg/h for up to 72 hours. This regimen resulted in a reduction of rebleeding from 22.5% to 6.7%, representing a number needed to treat (NNT) of 6 to prevent one person bleeding within 30 days. Subsequent studies using lower intravenous doses of omeprazole or

high-dose oral omeprazole also demonstrated a reduction in rebleeding rate. Further study is required to determine the optimum dose and schedule of proton pump inhibitors in UGIB. It seems reasonable, however, to treat patients with high-risk SRH with intravenous or high-dose oral proton pump inhibitors after endoscopic therapy has been administered.

ALGORITHM FOR MANAGEMENT OF NONVARICEAL UGIB



VARICEAL BLEED

Variceal bleeding is one of the dreaded complications of portal hypertension. Although its prognosis has improved over the last several decades, it still carries substantial mortality. Although most portal hypertensive bleeds result from the ruptured distal esophageal varices, bleeding from other sources such gastric varices, portal hypertensive gastropathy, and ectopic varices can lead to clinically significant bleeding. The following sections review management of acute variceal bleeding, prevention of rebleeding, bleeding from gastric varices and portal hypertensive gastropathy, and the prevention of first variceal bleeding.

MANAGEMENT OF ACUTE VARICEAL BLEEDING

The following guidelines optimizes the management of acute variceal bleed

1. Initial resuscitation
2. Early endoscopy
3. Endoscopic therapeutic procedures like Endoscopic variceal banding or sclerotherapy. (EVL slightly better than EST).
4. Vasoconstrictors like Octreotide , Somatostation, Terlipressin[88]. Studies have demonstrated the supremacy of Terlipressin over others.

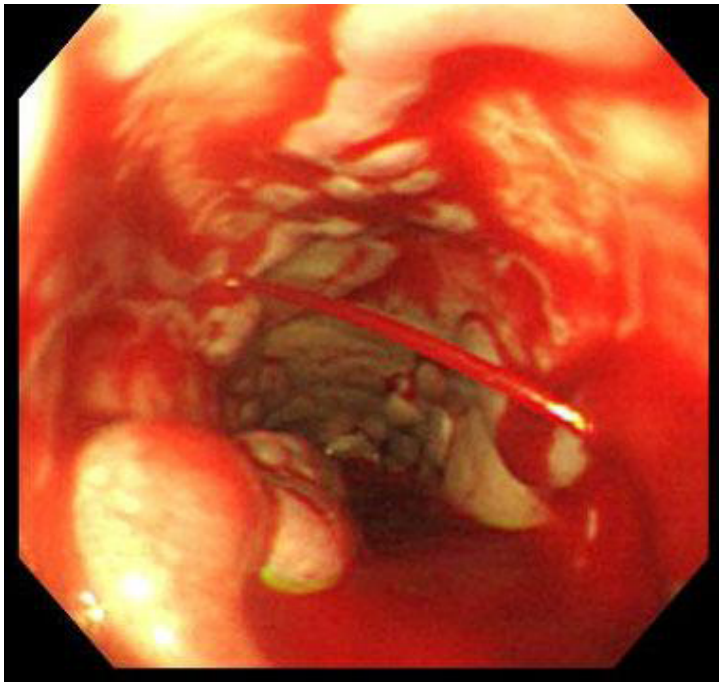


FIG 3: BLEEDING ESOPHAGEAL VARICES

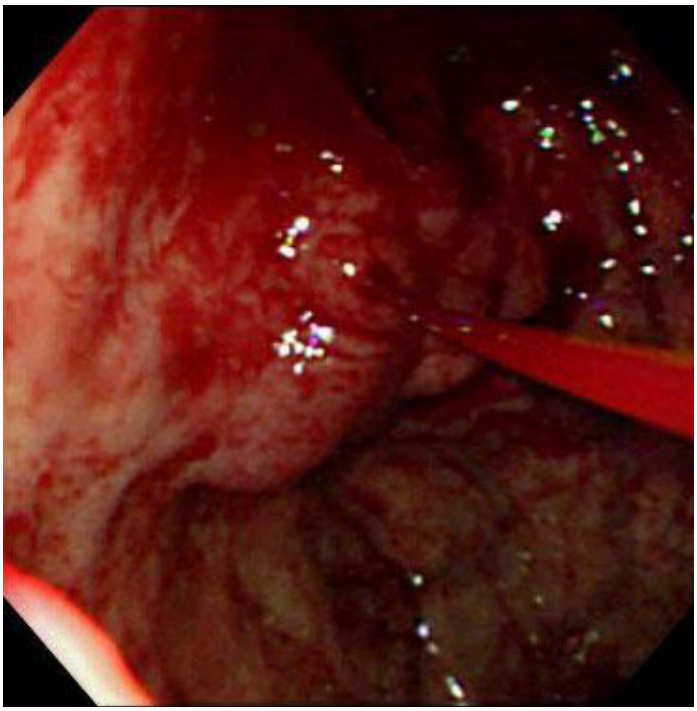


FIG 4: BLEEDING FUNDAL GASTRIC VARICES

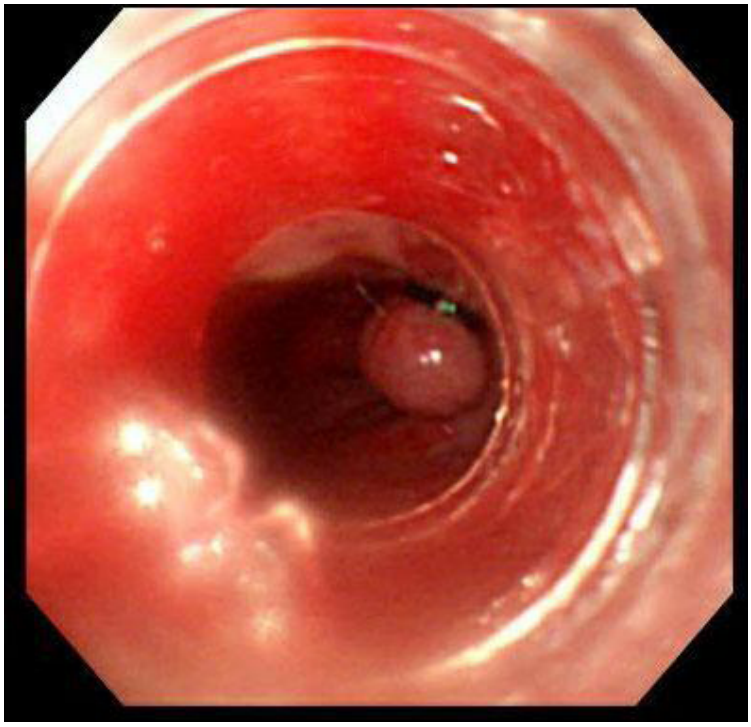


FIG 5: ENDOSCOPIC VARICEAL BANDING

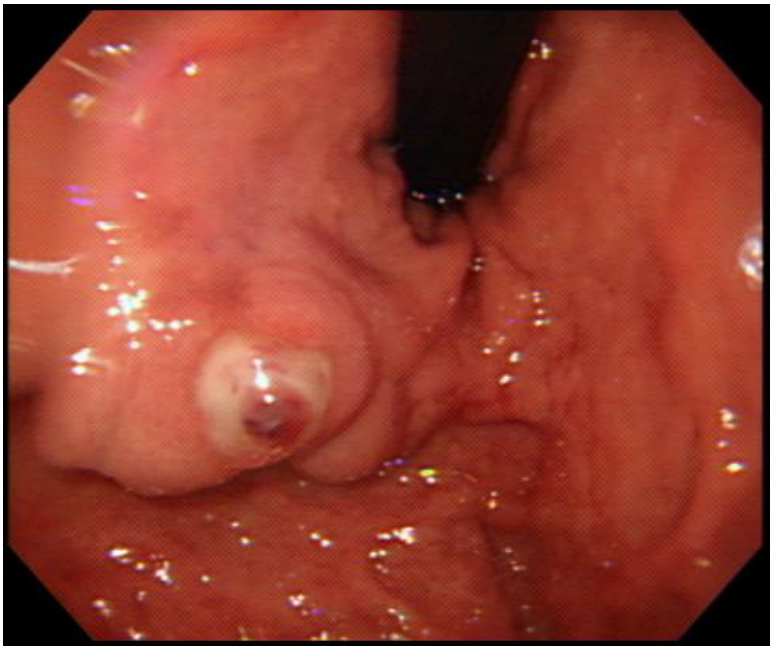
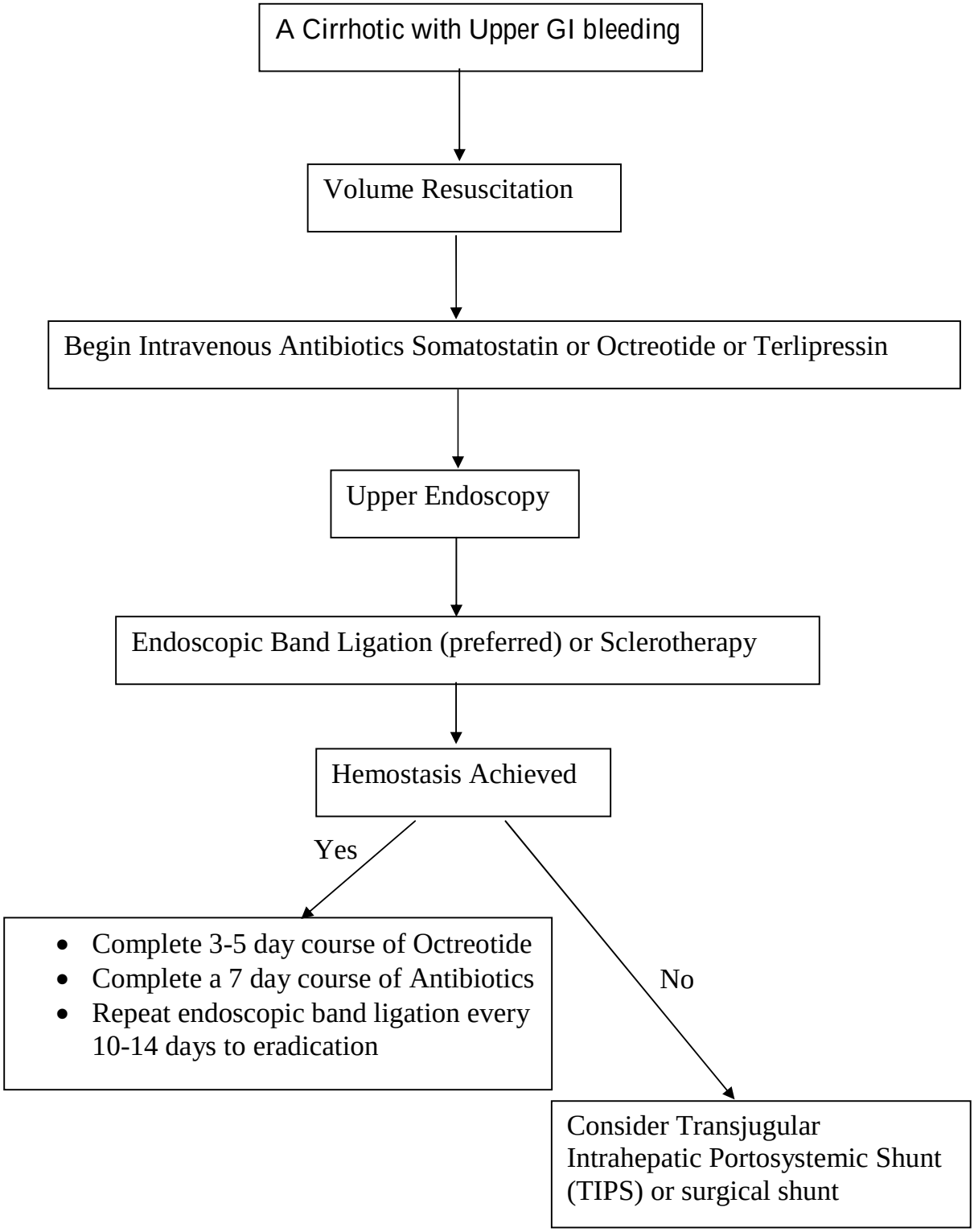


FIG 6: FUNDAL GASTRIC VARIX WITH EVIDENCE OF RECENT bleed

5. TIPS[89] (Trans arterial Intra hepatic Portovenous Stent) used as salvage for those patients who could not be treated by endotherapy or who rebleeds.
6. Shunt surgeries should be reserved as the last resort for refractory bleed

Algorithm for managing acute variceal bleed



PREVENTING RECURRENT VARICEAL BLEEDING

Without further therapy, once initial control is achieved, variceal bleeding recurs in two thirds of patients within 2 months. Factors associated with increased risk of recurrent bleeding include presence of active bleeding on initial endoscopy, large varices, severity of initial hemorrhage, decompensation, impaired renal function, presence of encephalopathy, and severe portal hypertension (as measured by the hepatic venous pressure gradient). Because of the high risk of recurrent hemorrhage, secondary prophylaxis should be initiated shortly after an episode of bleeding.

Pharmacologic Therapy

The main goal of pharmacologic is to significantly reduce portal hypertension, ideally to reduce the hepatic venous pressure gradient below12 mmHg, and to prevent recurrent bleeding. Therefore, the ideal way to adjust medical therapy would be to follow portal pressure as determined by the hepatic venous pressure gradient. The best time to measure portal pressure would be within the first month after a bleeding episode to determine which patients have severe portal hypertension and have the greatest risk of recurrent bleeding. More than a 20% reduction in portal pressure has been shown to significantly reduce the cumulative probability of recurrent bleeding from 28% to 4% in the first year and

from 39% to 9% at 2 years. Unfortunately, pressure measurements are expensive, invasive, and not readily available. Therefore, surrogate measures of portal pressure reduction commonly are used, such as a target heart rate of 55 beats per minute or a 25% reduction in the heart rate from baseline. Not all patients, however, are protected from bleeding. Recent studies have shown that despite adequate beta-blocker therapy, there are a percentage of patients that still have hepatic venous pressure gradients above 12 mmHg, which puts them at continued risk for variceal hemorrhage[90].

Several trials have demonstrated the efficacy of a nonselective beta-blocker compared with placebo in decreasing the risk of recurrent bleeding and improving survival. The addition of isorbide mononitrate (ISMN) to a betablocker regimen appears to further reduce the rate of rebleeding. In addition, recent studies have shown that combination pharmacologic therapy may be superior to sclerotherapy and band ligation. Incidence of rebleeding was 25% over an 18-month period with combination medical therapy compared with 53% for sclerotherapy in Child-Pugh class A or B cirrhotics these same investigators compared combination therapy with band ligation and demonstrated a reduction in bleeding frequency from 49% to 33%. The benefit of combined medical therapy, however, was realized mainly in patients who had Child-Pugh

class A or B cirrhosis. More recent studies comparing combination pharmacologic therapy with band ligation revealed conflicting results. Lo and colleagues observed that band ligation was superior to combination medical therapy. A study by Patch and colleagues observed that both treatment modalities were equivalent. Most likely the differences in results lie in study methods. Combination therapy may be superior to endoscopic therapy. The adverse effects of pharmacologic therapy, especially with combination pharmacologic medical therapy, however, can limit compliance.

Endoscopic therapy

Even though sclerotherapy has been shown to be effective in reducing recurrent variceal hemorrhage and appears to be equivalent to beta-blocker therapy, band ligation appears to have similar efficacy in decreasing recurrent bleeding, but fewer complications and higher survival rates[91]. Therefore, for endoscopic therapy to prevent rebleeding, band ligation should be considered the procedure of choice. Combination band ligation with pharmacologic therapy may be the ideal treatment modality. So far, there have been two published trials comparing band ligation alone with band ligation plus pharmacologic therapy. In a study by Lo and colleagues comparing band ligation alone with band ligation plus nadolol and sucralfate, rebleeding was reduced from 47% to 23%

with combination therapy. A more recent study by Pena and colleagues comparing band ligation alone with band ligation plus nadolol, demonstrated that the rebleeding rate was reduced from 38% to 14% with the combination group. In addition, postbanding ulcers are common, and significant bleeding from these ulcers occurs in 2% to 5% of cases. Varices rebleeding rates potentially can be reduced further by adding antiulcer therapy after endoscopic therapy. Shaheen and colleagues performed a study to evaluate the efficacy of proton pump inhibitor in treating postbanding ulcers in the setting of elective endoscopic band ligation. This was a randomized double-blinded, placebo-controlled trial. After elective endoscopic band ligation, subjects received either intravenous pantoprazole 40 mg followed by 40 mg oral pantoprazole daily for 9 days (n ¼ 22) or intravenous/oral placebo (n ¼ 22). There was no difference in the number of postbanding ulcers among the groups. Ulcers in the control group, however, were twice as large as the pantoprazole group (82 mm2 versus 37 mm2, $P < .01$). Two patients had postbanding ulcer bleeding; both were in the control group ($P > .05$). Because proton pump inhibitors are tolerated well and simple to administer, their use in this setting appears reasonable.

Surgical Shunt and Transjugular Intrahepatic Portosystemic Shunt

Portocaval or distal splenorenal shunts have been used in preventing recurrent variceal bleeding. A meta-analysis comparing distal splenorenal shunt with sclerotherapy found that shunt placement significantly reduced the rate of recurrent bleeding but also increased the incidence of encephalopathy and did not improve survival. Rebleeding after surgical shunts typically is caused by shunt thrombosis, which occurs usually within the first year. It is unusual for surgical shunts to thrombose beyond 1 year. Similarly, with TIPS compared with endoscopic therapy the rebleeding rate was significantly lower with TIPS, 19% versus 47%, but the incidence of encephalopathy was higher with TIPS, 34% versus 19%, with no difference in survival. Rosemurgy and colleagues compared TIPS with a surgically place H-graft shunt. This was a nonrandomized study of 132 patients. Rosemurgy and colleagues observed that the frequency of rebleeding was significantly less in the surgical group (3% versus 16%), and the patients who had TIPS required frequent interventions to maintain shunt patency. Thirty-day mortality rates, however, were higher in the surgical group, 43% versus 15%. Therefore, surgical shunts should be used to prevent rebleeding in patients who do not tolerate or are not compliant with medical therapy and have

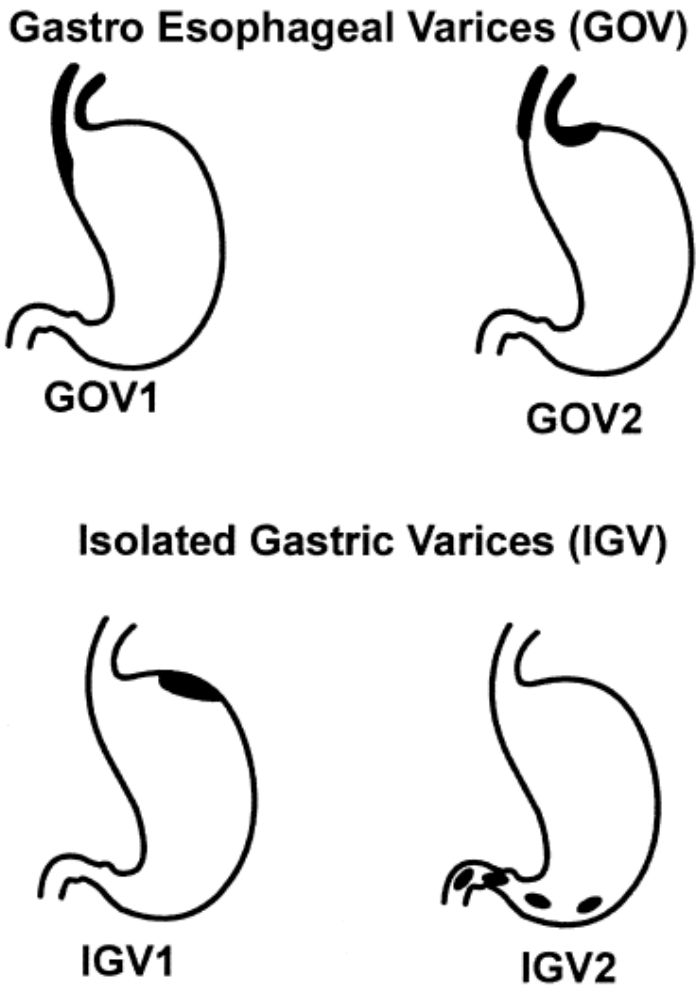
relatively preserved liver function. TIPS should be reserved for patients who have poor liver function and who have failed medical therapy.

BLEEDING FROM PORTAL HYPERTENSIVE GASTROPATHY

Portal hypertensive gastropathy (PHG) is the characteristic mosaic-like gastric mucosa with or without red spots; it is seen quite frequently in patients with both cirrhotic as well as noncirrhotic portal hypertension. Although the bleeding from PHG can be acute or chronic in nature, chronic bleeding presenting as iron deficiency anemia or occult blood in stool is far more frequent than acute bleeding. The specific treatment options for significant PHG include nonselective beta-blockers, endoscopic therapy, or TIPS or surgical shunts[92]. Nonselective beta-blockers have been shown to reduce the risk of bleeding in patients who have PHG. Endoscopic therapy in the form of cauterization (heater or bipolar probe or argon plasma coagulation) or injection sclerotherapy can be effective in patients who have acute bleeding caused by PHG. With argon plasma coagulation, especially if such endoscopic expertise is available locally. TIPS can reduce the bleeding from severe PHG effectively.

BLEEDING FROM GASTRIC VARICES

Gastric varices are rare but important sources for bleeding in patients who have portal hypertension. Gastric varices can be classified into gastro–esophagealvarices (GOV) or isolated gastric varices (IGV). GOV are classified further into GOV 1 (in continuity with esophageal varices and extend 2 to 5 cm below the gastroesophageal junction) or GOV 2 (esophageal varices extending into the fundus). IGV can be located in the fundus (IGV 1) or body/antrum (IGV 2). Gastric varices located in the gastric fundus (either GOV 2 or IGV 1) carry a greater risk of bleeding than those located in other parts of the stomach.



Potential treatments for gastric variceal bleeding include endoscopic (cyanoacrylate[93] or its derivatives or thrombin), radiological (TIPS or balloon-occluded retrograde transvenous obliteration)[94], and surgical (gastric devascularization and splenectomy, surgical shunts, or liver transplantation) modalities.

MATERIALS & METHODS

Materials and Methods

This study was done at Coimbatore Medical College Hospital, Coimbatore from March 2009 to November 2010 after getting approval by the Ethical committee.

Study Design:

Prospective Study

Inclusion Criteria:

100 patients admitted in medical, surgical and specialty departments of Coimbatore Medical College Hospital with history of Upper Gastrointestinal Bleed and in whom UGI scopy was performed were included in this study.

Exclusion Criteria:

- Children less than 12 years of age
- Pregnant women
- Hemodynamically unstable patients
- Patients with coagulopathies, epistaxis, or gum bleed

All patients were informed prior to the procedure about nature of procedure and a well informed consent was obtained.

Study Methodology:

A complete history was taken from all enrolled patients which included

1. No of episodes of hematemesis
2. Approximate quantity of bleed (total)
3. Presence of melena
4. History of previous Upper Gastrointestinal Bleed or treatment for Upper Gastrointestinal Bleed
5. History of intake of NSAIDs including aspirin and steroids
6. History of alcohol intake and smoking
7. History of prior surgery, organ transplantation

A thorough clinical general and systemic examination was done. The following factors were assessed

1. General condition and vital signs
2. Assessment of severity of blood loss (BP, Postural Hypotension, Tachycardia)
3. Assessing signs of portal hypertension (ascites, splenomegaly and jaundice)
4. Presence of hepatic encephalopathy

Laboratory tests:

The following lab investigations were done.

1. Hemoglobin, Total Count, Differential Count
2. Packed Cell Volume
3. Blood Urea and Serum Creatinine
4. Blood grouping and Rh typing
5. Coagulation profile
6. Liver function test (Serum bilirubin, SGOT, SGPT, Alkaline phosphatase, Serum proteins)

Upper Gastrointestinal Endoscopy:

Upper Gastrointestinal endoscopy was done in the Department of Gastroenterology, Coimbatore Medical College Hospital using Pentex EG291C endoscope after eight hours of fasting to directly visualize the mucosa of the esophagus, stomach and duodenum.

The endoscopic stigmata of active or recent hemorrhage and endoscopic prognostic features like number of ulcers, site and location of ulcers, size of ulcers, bleeding or not healing or not, clean base of the ulcer or adherent blood clot, oozing of blood from the ulcer base and visible blood vessel were studied. The site, grading of varices were studied and search for rare causes for UGI bleed were made.

Endoscopic interventions like injection therapy, Endoscopic sclerotherapy, EVL were done as needed.

All patients were treated with resuscitative measures, transfusions, vasoconstrictors, PPI, H2RI and antibiotics as needed.

All data were tabulated.

Statistical Analysis :

Done with Chi-square with ANNOVA

OBSERVATIONS & RESULTS

OBSERVATIONS AND RESULTS

Upper gastrointestinal bleed is a common medical emergency. Coimbatore medical college hospital is a tertiary care institution which caters to three districts and treats sizeable number of patients with upper gastrointestinal bleed. 100 cases that presented with UGI bleed and underwent UGI scopy were evaluated for the study.

Sex Distribution:

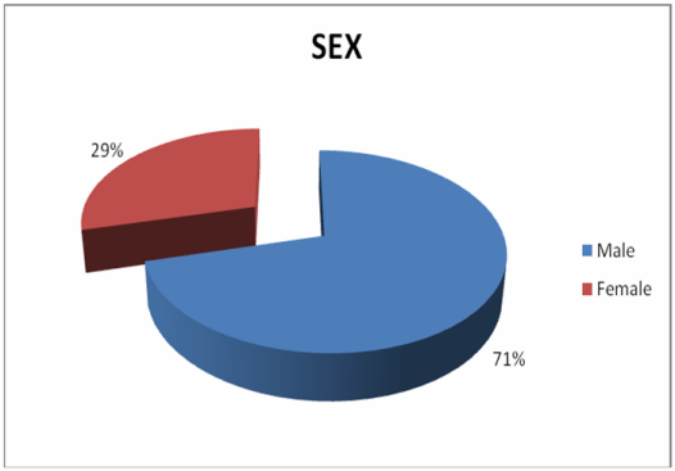
In this study of 100 cases of UGI Bleed the sex distribution is observed as

Table 5.1 :

Gender Distribution

Sex	No. of Cases	Total No. of Cases	Percentage (%)
Male	71	100	71%
Female	29	100	29%

Figure: 5.1



Males were predominantly affected with UGI bleed (71%).

Age Distribution:

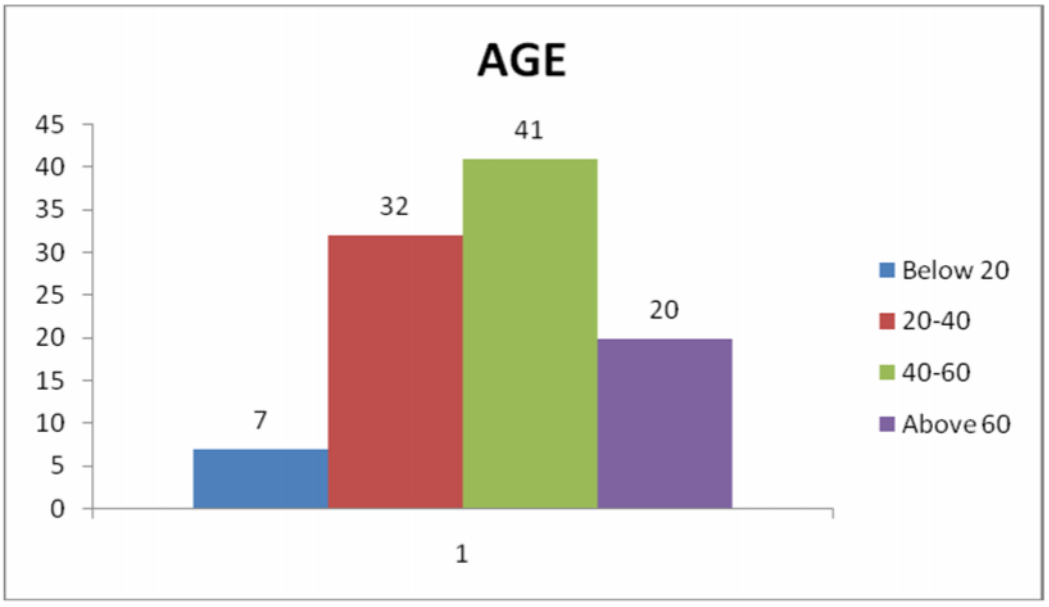
In this study of 100 cases of upper Gastrointestinal bleed the age distribution observed is as follows

Table 5.2

Age wise Distribution

Age in years	No. of Cases	Total No. of Cases	Percentage (%)
< 20	7	100	7%
20-40	32	100	32%
40-60	41	100	41%
>60	20	100	20%

Figure 5.2:



Predominant age group is 40-60 years, accounting for 41 %.

Hemoglobin:

We assorted the patients admitted with upper gastrointestinal bleed into two groups based upon the hemoglobin values in grams/dl.

The results are given below.

Table 5.3

Hemoglobin at Initial Presentation

Hb (grams /dl)	No. of cases	Total No. of cases	No. of Deaths
<8	34	100	9
> 8	66	100	0

Vital Parameters:

Pulse, blood pressure and conscious level play an important role in prognostication. The results are as follows

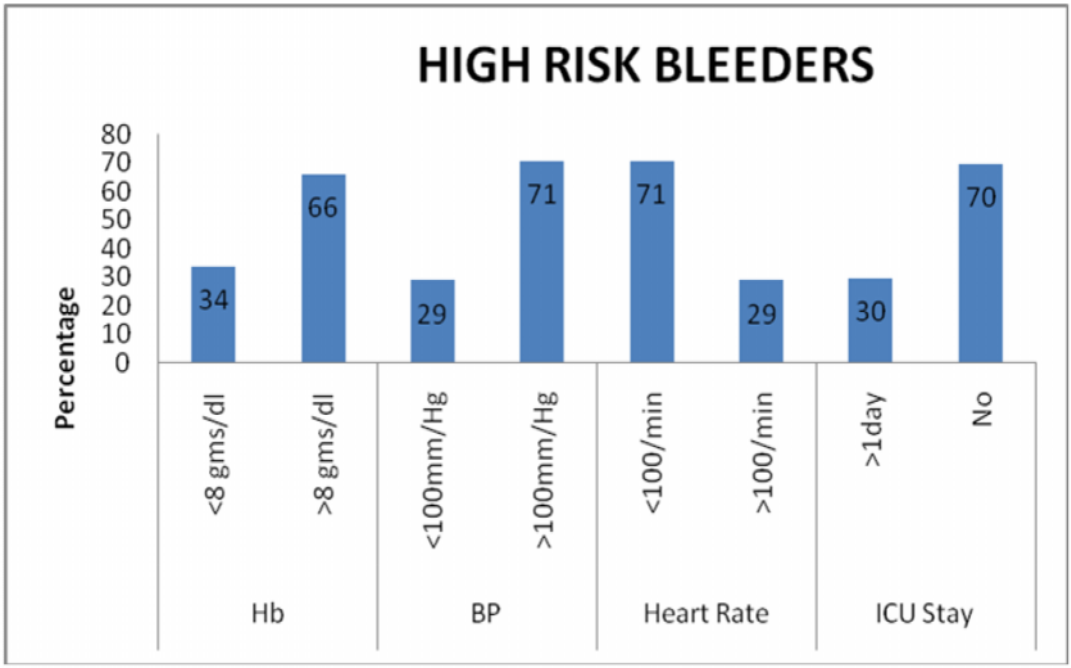
Table 5.4. Vitals

Parameter	Grade	No. of Patients	Total No. of cases
Pulse	<100	82	100
	>100	18	100
Blood pressure	< 100	29	100
	>100	71	100

ICU Stay

Of the 100 cases in whom endoscopy was done 30 patients were admitted in Intensive Care Unit and there were 9 deaths in total. All deaths occurred in patients admitted in ICU.

Fig. 5.3.



Tachycardia, Hypotension, ICU stay and Encephalopathy are high risk features.

History:

- ❖ Out of 100 patients, 48 (48%) patients were alcoholics
- ❖ 57 (57%) patients were found to be smokers
- ❖ History of NSAID intake was observed in 17 (17%) cases.

- ❖ Previous history of UGI bleed was present in 11 (11%) cases.

Comorbidities

Comorbidities are major determinants of adverse outcomes in UGI bleed according to several studies, but in our study comorbidities did not affect the outcome significantly probably due to lesser number of elderly patients and lower incidence of comorbid illness in our study.

In our study, 17 patients had ascites, 3 had hepatic encephalopathy, 17 patients had hypertension and CAD and renal failure was noted in 5 cases.

Endoscopic Findings:

In this study in Upper Gastrointestinal Bleed the frequency of clinical presentations observed are as follows

Table 5.5.

Endoscopic Findings in 100 cases of UGIB.

Nature of lesion	No. of cases	Percentage (%)
Duodenal Ulcer	25	25%
Gastric Ulcer	4	4%
Esophageal Varices Gr-I	2	2%
Esophageal Varices Gr-II	5	5%
Esophageal Varices Gr-III	13	13%
Esophageal Varices + Fundal Varices	3	3%
Gastric erosions	28	28%
Duodenitis	3	3%
Mallory Weiss	12	12%
Esophageal Ulcer	1	1%
Esophagitis	3	3%
Gastric Malignancy	4	4%

Peptic ulcer was the most common cause of upper gastrointestinal bleed, with gastric erosion (28 %) being the most frequent lesion. It was found that duodenal ulcer was the second most common lesion contributing 25% of UGIB. Variceal bleeding was noted in 20% of the patients.

Table 5.6.

FORREST GRADING OF PEPTIC ULCER BLEED

Lesion	No of Cases
Active bleeding	7
NBVV	2
Adherent clot	10
Clean base or pigmented ulcer	19

Table -5.7.

VARICEAL FINDINGS

Lesion		No of patients
Grade	Mild	2
	Moderate	5
	Severe	13
Column	Mild	2
	Moderate	5
	Severe	13
Gastric varices	Present	3
PHT Gastropathy	Present	6
Red Colour signs	Present	11

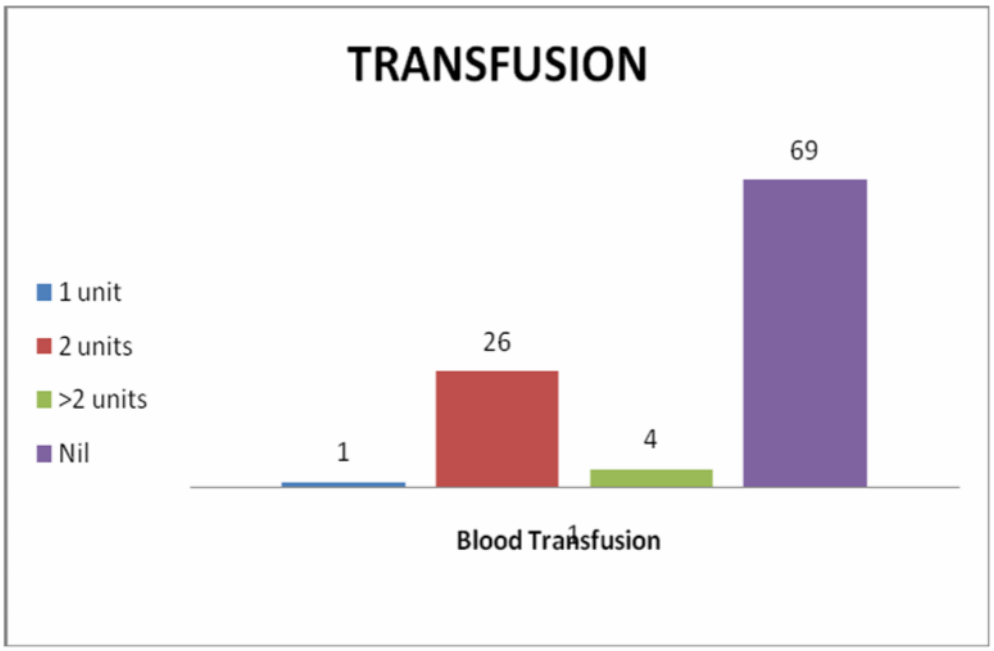
High grade varices, red colour lesions, bluish varices are high risk features for rebleed and mortality. 13 patients had high grade varices.

Blood Transfusions:

Out of 100 patients, 31 patients required blood transfusion and out of them 4(4%) patients required more than 2 units of transfusions. Blood transfusion was not required in 69(69%) of cases

Fig 5.4.

Blood Transfusion Requirement



Discussion

DISCUSSION

After enumerating the etiologies of UGIB, peptic ulcer constitutes the most common cause, out of which duodenal ulcer constitutes 25%, gastric erosions 28%, gastric ulcer 4%, duodenitis3% and esophageal ulcer 1%.

This data correlates well with the UK registry of UGIB (Audit committee) which was published in BMJ by Rockall TA et al. The Canadian registry also has similar observations.

Malignancy accounted for 4% of cases and varices constituted 20% of non ulcer bleed.

UGI scopy was the diagnostic modality of choice and done in all cases. 74% of UGI scopy were done within 24 hours. Endoscopic intervention were done as appropriate and sclerotherapy was the most preferred procedure than EVL for esophageal varices. This choice was based on local preference, expertise and simplicity. Adrenaline injection was the preferred endotherapy for ulcer bleed.

Compared data on the choice, EVL is the most common procedure worldwide and widely accepted for esophageal variceal bleed. Head to head trials comparing EST and EVL showed superiority of EVL over

EST but visibility of the endoscopist after a torrential bleed is of concern during EVL (Laine L, Cook D et al). Total number of death is 9 (9%).

We have formulated diagnostic criteria for major bleed as follows:

1. Death of the patient
2. Who had transfusion requirement of more than 2 units
3. Who had ICU stay more than 1 day
4. Hospital stay more than 3 days.

We have chosen a few patient parameters as potential risk factors for early prediction of major UGIB. The parameters considered are age, BP, HR, Hb at presentation,creatinine level, timing of endoscopy, endoscopic diagnosis and endotherapy.

Using statistical analysis the following were observed :

Age was considered as an important predictor of death. Age > 60 is associated with increased mortality ($p<0.001$). Age is not a predictor of increased ICU stay ($P < 0.102$), increased transfusion requirement ($P<0.0305$) or hospital stay ($p<0.02$).

This is compared with Rockall et al which has similar mortality in > 60 years age group. Mortality increased with advancement of age (Blatchford et al)

Blood pressure at presentation is found to have important predictor of death and major bleed. BP < 100/70 is associated with increased death

(P <0.001), increased hospital stay (P < 0.001) and increased transfusion requirement (P<0.001).

Similar way cold peripheries (Shock) at presentation predicts mortality, increased ICU stay, hospital stay and transfusion requirement (P<0.001).

Study of Longstreth GF 1997 indicated that outcome of patient who presented with shock had higher mortality and constituted about 17 % of all bleeds.

Heart rate >100 correlated well with mortality (P<0.001), ICU stay (P<0.001) and hospital stay (P<0.001).

Hb < 8gms/dl predicted the need for increased transfusion (P<0.001), ICU stay (P=0), hospital stay (P=0) and death (P=0)

Creatinine >2mg/dl correlated well with death, ICU stay, transfusion and hospital stay (P =0)

Presence of coronary artery disease is surprisingly neither a predictor factor of mortality (P<0.171), ICU stay (P<0.954), transfusion (P<0.799) nor hospital stay (P<0.481)

Presence of comorbidities like HT, CRF, arthritis, alcoholism or smoking did not predict death of major bleed. This is in contrast to study by Rockall which predicted high mortality for patients with comorbid

illness. The discrepancy occurred because of skewed patients population towards younger age and low incidence of comorbid illness in our study.

Timing of endoscopy and its possible improved outcome is a major controversy in management of UGIB.

Various studies have produced variable results. In general UGI scopy performed <24hrs did not have any added advantage in reducing mortality or morbidity (Splejel BM et al).

Optimal timing of UGI scopy is put variably by different authors. But the consensus statement put it at < 24 hrs (Stering committee Rockall et al).

Our series had 95 % of UGI done within 24 hrs. 78 % of patients who died had UGI done <24 hrs. 22 % had UGI done after 24hrs probably due to continued hemodynamic instability and resuscitation. The mortality in these subgroup is statistically significant to early UGI scopy group (P<0.004).

The timing of UGI correlated well with ICU stay and hospital stay has expected. Patient who underwent endoscopy < 24hrs had good recovery despite major bleed (P<0.004)

Endoscopic diagnosis of etiology emerged as the strongest predictor of mortality. Diagnosis of esophageal varices, bleeding peptic

ulcer contributed 98 % of all mortality signifying the importance ($P < 0.001$).

Endotherapy with sclerosant, adrenalin conveyed mixed results when correlated with mortality. Patient needing endotherapy were very sick contributing to overall mortality ($P<0.001$) but 69 % (16/23) of patient were salvaged by endoscopy ($P<0.001$).

Conclusions

CONCLUSION

1. 30% of the patients with UGIB had high mortality and morbidity. (Major UGIB)
2. Strong predictors of major UGIB are Age>60, HR>100, BP<100/70, Hb < 8gms/dl, Creatinine>2mgs/dl and shock at presentation.
3. Endoscopy is the most valuable investigation for diagnosis. UGI scopy done < 24 hrs predicted well with improved outcome.
4. Endoscopic diagnosis like esophageal varices, bleeding peptic ulcer carried high mortality.
5. Endoscopic therapy (injection for ulcer bleed, sclerotherapy for varices) salvaged 69 % of major bleed and improved prognosis.

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Annexures

INFORMED CONSENT

Department of General Medicine

Coimbatore Medical College, Coimbatore

Principal Investigator: Dr Anand Kumar

Research Guide: Dr. M. Raveendran

Organization: Department of General Medicine

Informed consent

I have been invited to participate in the research project titled "Clinical Vs Endoscopic Correlation of Upper Gastro Intestinal Bleeding"

I understand it will involve answering a set of questionare, undergo physical examination and to undergo Upper GI endoscopy.

I am aware of the procedure of endoscopy which may be associated with discomfort and carries very minimal risk of adverse complications. I am also aware that it may necessitate interventions through endoscope as the case may warrant.

Also I give consent to utilize my personal details for study purpose and can be contacted if necessary.

I am aware that I have the right to withdraw at any time which will not affect my medical care.

Name of the participant:

Signature:

Date:

PROFORMA

Study No :				
Name:		Age:		Sex:
IP No:				ward:
Address :				Contact No:
History:				
UGI bleed	Hematemesis		Melena	Both
Date	No of Times			
Approx qty of Bleed				
Prior Hospitalizations	Details			
Blood Transfusion				
H/O Alcohol	Details			
H/O NSAIDs	Details			
H/O Smoking	Details			
H/O Aspirin	Details			
K/c of Liver disease		PUD		Others
DM	SHT IHD	CKD	Others	
Surgery in the past				
Jaundice in the past	Details			
Previous endoscopy				
After admission:				
HR	BP	SpO2		
No of blood transfusion required				
Ryles tube aspirate	Clear		Blood	
Basic investigations				
Hb	PCV Platelets	Urae	Creatinine	
ECG	CXR USG			
Endoscopy				
Performed after within 24hrs	> 24 hrs			
Findings at endoscopy				
Site of bleed				
Nature of the bleed				
Forrest grading for PUD bleed				
Paquet grade of Variceal bleed				
Intervention done				

LIST OF ABBREVIATIONS

- UGIB - Upper Gastro Intestinal Bleeding
- NSAID - Non Steroidal Anti-Inflammatory Drugs
- H.pylori - Helicobacter Pylori
- NVUGIB - Non Variceal Upper Gastro Instestinal Bleeding
- HVPG - Hepatic Venous Pressure Gradient
- CTP - Child Turcotte Pugh
- EVL - Endo Variceal Ligation
- EST - Endoscopic Sclero Therapy
- SRH - Stigmata of Recent Hemorrhage
- NBVV - Non Bleeding Visible Vessel
- HPC - Heater Probe thermo Coagulation
- TIPS - Transjugular Intrahepatic Porto Systemic shunt
- PHG - Portal Hypertensive Gastropathy
- GOV - Gastro-esophageal Varices
- IGV - Isolated Gastric Varices
- PPI - Proton Pump Inhibitor.
- ITU – Intensive Therapy Unit
- HPU – High Dependency Unit.

S.No	Patient Name	Endoscopy No	Age	SEX	Malena Alone	Melena and Haemetemesis	Findings	BP	COLD PERIPHERIES	heart rate	Hb	Urea	Creatinine	ICU Stay	Transfusion	Death	CAD	HT	chronic Renal Failure	Arthritis	PCV	Hospital Stay	UGI Done After	Interventions	Renal Transplant
1	Subramani	262	35	M	N	Y	Duodenal ulcer	100/70	N	88	12	40	O.8	N	N	N	N	N	N	N	36	2	>24hrs	no	N
2	Kandasamy	275	66	M	N	Y	Duodenal ulcer	70/50	Y	106	6.3	60	3.1	>1	2	N	Y	Y	N	Y	21	2	<24hrs	adrenaline	N
3	Ragavan	283	27	M	N	Y	Gastric erosions	110/70	N	76	14	36	0.8	N	N	N	N	N	N	N	42	2	>24hrs	no	N
4	Rangasamy	284	65	M	N	Y	Grade III Oesophageal varices + fundal varices	70/40	Y	112	4.6	54	2.5	>1	>2	Y	N	N	N	N	15	3	<24hrs	Sclero	N
5	Dhara	297	17	F	N	Y	Duodenal ulcer	80/60	Y	118	6	52	2.3	>1	2	N	N	N	N	N	18	2	<24hrs	adrenaline	N
6	Jothi	311	30	F	N	Y	Duodenal ulcer	80/62	Y	110	5.6	46	1.7	>1	2	N	N	N	N	N	18	2	<24hrs	adrenaline	N
7	Madhi Antony	318	48	M	N	Y	Grade III Oesophageal Varices	80/60	Y	108	4.2	58	2.9	>1	2	N	N	N	N	N	16	4	<24hrs	sclero	N
8	Saraswathi	320	45	F	Y	N	Gastric ulcer	120/80	N	78	10.4	36	0.7	N	N	N	N	N	N	Y	33	2	>24hrs	no	N
9	Suganya	324	18	F	N	Y	Mallory - Weiss tear	110/80	N	88	10.8	32	0.8	N	N	N	N	N	N	N	33	2	>24hrs	no	N
10	Lakshmi	343	42	F	N	Y	Duodenal ulcer	68/46	Y	106	4.8	60	3.1	>1	2	N	N	N	N	Y	16	2	<24hrs	adrenaline	N
11	Selvi	361	25	F	N	Y	Mallory - Weiss tear	114/76	N	84	10.6	28	0.7	N	N	N	N	N	N	N	33	2	>24hrs	no	N
12	Parvathy	368	85	F	N	Y	gastric erosions	80/60	Y	118	6.8	64	3.5	>1	2	Y	Y	Y	Y	Y	21	3	<24hrs	no	N
13	Veerasamy	369	61	M	Y	N	gastric erosions	120/74	N	88	11	26	0.6	N	N	N	N	N	N	N	33	2	>24hrs	no	N
14	Agnes	383	13	F	Y	N	gastric erosions	110/76	N	98	12	26	0.8	N	N	N	N	N	N	N	36	3	>24hrs	no	N
15	Babu	384	45	M	N	Y	Grade II Oesophageal Varices	80/60	Y	120	6.2	52	2.3	>1	2	N	N	N	N	N	18	3	<24hrs	Sclero	N
16	Moorthy	385	42	M	N	Y	Grade III Oesophgeal varices Narrowed Pylorus	80/60	Y	116	4.1	59	3	>1	2	N	N	N	N	N	16	4	<24hrs	Sclero	N
17	Murugesan	388	36	M	Y	N	gastric erosions	110/80	N	88	13.2	28	0.6	N	N	N	N	N	N	N	39	2	>24hrs	no	N
18	Murugan	394	33	M	N	Y	Mallory - Weiss tear	114/80	N	80	13.5	32	0.6	N	N	N	N	N	N	N	39	2	>24hrs	no	N

19	Natraj	404	50	M	Y	N	gastric erosions	120/78	N	78	13.6	36	0.8	N	N	N	Y	Y	N	Y	34	2	>24hrs	no	N
20	Shajagan	405	25	M	N	Y	gastric erosions	110/70	N	76	13.4	32	0.9	N	N	N	N	N	N	N	40	2	>24hrs	no	N
21	Darmaraj	407	38	M	N	Y	Duodenal ulcer	100/70	N	86	12.6	30	1	N	N	N	N	N	N	N	36	2	>24hrs	no	N
22	Kalamani	413	22	F	N	Y	gastric erosions	112/80	N	84	10.6	27	0.8	N	N	N	N	N	N	N	30	3	>24hrs	no	Y
23	Senthil Kumar	416	41	M	N	Y	gastric erosions	120/78	N	82	12	31	0.8	N	N	N	N	N	N	N	36	2	>24hrs	no	N
24	Selva Raj	427	28	M	N	Y	Duodenal ulcer	110/78	N	76	12.6	36	0.9	N	N	N	N	N	N	N	39	2	>24hrs	no	N
25	Karupusamy	454	19	M	Y	N	Duodenal ulcer	130/80	N	90	13	30	0.7	N	N	N	N	N	N	N	42	2	>24hrs	no	N
26	Selvakumar	455	27	M	N	Y	Esophagitis	110/78	N	94	12.4	24	0.7	N	N	N	N	N	N	N	35	2	>24hrs	no	N
27	Iqbal	457	47	M	N	Y	Duodenal ulcer	70/40	Y	114	4.7	64	3.5	>1	2	Y	Y	Y	N	Y	15	2	<24hrs	adrenaline	N
28	Mahalingam	460	62	M	N	Y	gastric erosions	112/80	N	78	12	32	1.1	N	N	N	Y	Y	N	Y	36	3	>24hrs	no	N
29	Vasanthan	462	40	M	N	Y	grade III Oesophageal varices	80/60	Y	102	4	57	2.8	>1	2	N	N	N	N	N	14	3	<24hrs	Sclero	N
30	Kanagaraj	466	20	M	N	Y	Mallory - Weiss tear	112/76	N	84	12.4	27	0.8	N	N	N	N	N	N	N	36	4	>24hrs	no	N
31	Kumarasamy	469	38	M	N	Y	grade III Oesophageal varices	80/60	Y	120	3.9	55	2.6	>1	2	N	N	N	N	N	14	4	<24hrs	Sclero	N
32	Kannan	474	38	M	Y	N	gastric erosions	120/78	N	82	12.6	24	0.8	N	N	N	N	N	N	N	36	2	>24hrs	no	N
33	Laxmanan	479	48	M	N	Y	Grade I Oesophageal varices	80/60	Y	118	6.7	61	3.2	>1	2	N	Y	Y	N	Y	21	3	<24hrs	no	N
34	Arusamy	487	70	M	N	Y	gastric ulcer	110/70	N	78	12.6	32	0.7	N	N	N	N	N	N	Y	38	2	>24hrs	no	N
35	Revathi	489	16	F	N	Y	Duodenal ulcer	100/70	N	80	11.2	23	0.8	N	N	N	N	N	N	N	33	1	>24hrs	no	N
36	Baskar	492	25	M	N	Y	Duodenal ulcer	106/76	N	98	12.6	37	0.8	N	N	N	N	N	N	N	38	2	>24hrs	no	N
37	Ravi	496	42	M	N	Y	gastric erosions	110/76	N	78	12.8	38	0.7	N	N	N	N	N	N	N	39	2	>24hrs	no	N
38	Jagadeesh	498	45	M	N	Y	grade III Oesophageal varices + Fundal Varices	70/40	Y	116	5.5	63	3.4	>1	2	Y	N	N	N	N	18	3	>24hrs	sclero	N
39	ponnan	500	50	M	N	Y	Scarred Oesophageal varices	80/60	Y	114	6.7	54	2.5	>1	2	N	N	N	N	N	21	3	<24hrs	no	N
40	Loganathan	505	45	M	N	Y	Duodenal ulcer	110/74	N	84	11.6	40	1.2	N	N	N	N	N	N	N	35	2	>24hrs	no	N

41	Arokyaraj	507	61	M	N	Y	Duodenal ulcer	100/70	N	88	13.2	28	0.8	N	N	N	Y	Y	N	Y	39	2	>24hrs	no	N
42	Radha Mani	512	40	F	N	Y	Duodenal ulcer	70/56	Y	124	4.6	53	2.4	>1	2	N	N	N	N	Y	15	1	<24hrs	adrenaline	N
43	Sitarasan	519	44	M	N	Y	Grade II Oesophageal varices	80/60	Y	112	5.6	50	1.6	>1	2	N	N	N	N	Y	18	4	>24hrs	evl	N
44	Ajith Kumar	520	13	M	N	Y	Mallory - Weiss tear	114/74	N	94	13.6	22	0.7	N	N	N	N	N	N	N	45	2	>24hrs	no	N
45	Nagaraj	532	73	M	Y	N	Gastric malignancy	106/78	N	98	7.5	40	3	N	N	N	N	N	N	N	24	4	>24hrs	no	N
46	Palanisamy	536	50	M	N	Y	Grade III/IV Columns Oesophageal varices	70/40	Y	110	6.9	68	3.7	>1	2	Y	N	Y	N	N	21	4	<24hrs	sclero	N
47	Mahadevan	538	57	M	N	Y	grade III Oesophageal varices + fundal varices	70/40	Y	108	4.3	61	3.2	>1	>2	Y	N	Y	N	N	15	4	<24hrs	sclero	N
48	Arumugam	539	35	M	N	Y	Grade III Oesophageal varices with 3 Columns	80/60	Y	106	3.8	50	2.1	>1	2	N	N	N	N	N	14	3	<24hrs	evl	N
49	Hariharan	540	38	M	Y	N	Duodenitis	120/78	N	90	13.6	34	0.9	N	N	N	N	N	N	N	45	1	>24hrs	no	N
50	Poovathal	547	45	F	N	Y	gastric erosions	118/78	N	88	10.4	26	0.8	N	N	N	Y	Y	N	N	33	3	>24hrs	no	N
51	Tulasiammal	555	50	F	N	Y	Duodenal ulcer	70/40	Y	116	3.9	59	3	>1	>2	Y	Y	Y	N	Y	14	2	<24hrs	adrenaline	N
52	Valliammal	563	42	F	N	Y	Duodenal ulcer	110/76	N	82	7.5	34	0.6	N	N	N	N	N	N	N	24	2	>24hrs	no	N
53	Muthu	574	36	M	Y	N	gastric erosions	120/80	N	78	12.6	26	0.8	N	N	N	Y	Y	N	N	38	2	>24hrs	no	N
54	Masananan	575	48	M	N	Y	gastric ulcer	124/82	N	74	12.6	36	0.9	N	N	N	N	N	N	N	38	2	>24hrs	no	N
55	Devasagayam	579	45	M	N	Y	Grade I esophageal varices	80/60	Y	112	6.3	55	2.6	>1	2	N	N	Y	N	Y	21	5	<24hrs	no	N
56	Palanal	587	37	F	N	Y	Esophagitis	110/76	N	76	10.4	32	0.9	N	N	N	N	N	N	N	34	2	>24hrs	no	N
57	Rageswari	590	30	F	N	Y	Mallory - Weiss tear	126/78	N	80	10.6	28	0.9	N	N	N	N	N	N	N	30	4	>24hrs	no	N
58	Ravi	593	45	M	N	Y	Mallory - Weiss tear	114/74	N	88	9.8	28	0.9	N	N	N	N	N	N	N	28	5	>24hrs	no	N
59	Abdul Kutty	591	50	M	N	Y	gastric erosions	110/70	N	94	12.6	28	0.7	N	N	N	Y	Y	N	N	36	2	>24hrs	no	N
60	Srinivasan	597	60	M	N	Y	Grade II oesophageal varices	80/60	Y	120	5.8	56	2.7	>1	2	N	N	Y	Y	Y	36	4	<24hrs	Sclero	N
61	Renganathan	601	46	M	N	Y	GradeI oesophageal varices	80/60	Y	108	5.2	50	1.9	>1	2	N	N	N	N	N	18	3	<24hrs	no	N
62	Pechimuthu	602	32	M	N	Y	Duodenal ulcer	116/78	N	84	13.2	28	0.8	N	N	N	N	N	N	N	15	1	>24hrs	no	N

63	Kaleeswari	2	43	F	N	Y	Mallory - Weiss tear	120/78	N	78	10.2	28	0.7	N	N	N	N	N	N	N	39	6	>24hrs	no	N
64	Sivagami	18	60	F	N	Y	Gastric malignancy	80/60	Y	104	6.1	42	1.4	>1	2	N	N	N	N	N	30	6	<24hrs	no	N
65	Elangovan	37	27	M	N	Y	gastric erosions	120/76	N	82	10.4	23	0.6	N	N	N	N	N	N	N	18	3	>24hrs	no	N
66	Shanthamani	41	44	F	Y	N	Duodenal ulcer	126/78	N	88	12.4	26	0.8	N	N	N	N	N	N	N	30	2	>24hrs	no	N
67	Vasanthi	43	32	F	Y	N	Duodenitis	110/70	N	94	10.4	32	0.8	N	N	N	N	N	N	N	36	1	>24hrs	no	N
68	Susammal	48	55	F	N	Y	Grade lll oesophagial varices	100/70	N	98	6.8	48	1.7	>1	>2	Y	N	N	N	Y	30	4	<24hrs	Sclero	N
69	Selvi	50	30	F	Y	N	gastric erosions	124/76	N	90	10.2	26	0.8	N	N	N	N	N	N	N	21	3	>24hrs	no	N
70	Subramani	78	43	M	N	Y	gastric erosions	120/80	N	86	11.4	33	0.7	N	N	N	N	N	N	N	30	3	>24hrs	no	N
71	Chinna Durai	80	22	F	N	Y	Duodenal ulcer	110/70	N	90	10.4	26	0.9	N	N	N	N	N	N	N	33	2	>24hrs	no	N
72	Shanmugam	82	60	M	Y	N	gastric erosions	130/80	N	84	10	26	0.8	N	N	N	Y	Y	Y	N	30	2	>24hrs	no	N
73	Arumugam	83	45	M	N	Y	Grade lll oesophagial varices	80/60	Y	116	3.7	50	2.1	>1	2	N	N	N	N	N	30	4	<24hrs	Sclero	N
74	Chinna Mani	110	70	M	N	Y	Grade lll oesophagial varices	80/60	Y	112	5.2	60	3.1	>1	2	N	N	N	N	N	14	4	<24hrs	Sclero	N
75	Karuthammal	112	70	F	N	Y	gastric erosions	110/70	N	82	10.6	36	0.8	N	1	N	Y	Y	N	Y	15	3	>24hrs	no	N
76	Subramani	113	73	M	N	Y	gastric erosions	124/76	N	86	12.2	34	0.9	N	N	N	Y	Y	N	Y	33	2	>24hrs	no	N
77	Rajesh	121	40	M	N	Y	gastric erosions	110/70	N	90	13.2	32	0.8	N	N	N	N	N	N	N	36	3	>24hrs	no	N
78	Murugathal	126	40	F	N	Y	Duodenal ulcer	100/70	N	94	10.4	27	0.7	N	N	N	N	N	N	N	39	2	>24hrs	no	N
79	Sankar	131	30	M	Y	N	gastric erosions	124/82	N	78	12.4	29	0.8	N	N	N	N	N	N	N	30	2	>24hrs	no	N
80	Kalamani	140	30	F	Y	N	Duodenitis	106/76	N	74	10.6	32	0.9	N	N	N	N	N	N	N	36	1	>24hrs	no	N
81	Raju	161	82	M	N	Y	gastric ulcer	108/76	N	98	13.4	28	0.7	N	N	N	N	N	N	Y	39	3	>24hrs	no	N
82	Thulasimani	178	39	F	N	Y	Duodenal ulcer	120/80	N	90	10.4	35	0.9	N	N	N	N	N	N	N	30	2	>24hrs	no	N
83	Beer Mohammed	182	61	M	N	Y	Mallory - Weiss tear	110/76	N	84	12.6	38	0.9	N	N	N	N	N	N	N	37	2	>24hrs	no	N
84	Rajendran	184	28	M	N	Y	Duodenal ulcer	106/76	N	78	13.6	28	0.7	N	N	N	N	N	N	N	39	2	>24hrs	no	N

85	Guru Moorthy	191	45	M	N	Y	gastric erosions	124/82	N	90	13.8	88	4.6	N	N	N	Y	Y	N	N	41	2	>24hrs	no	N
86	Palanisamy	199	41	M	N	Y	Duodenal ulcer	110/70	N	96	12.4	36	0.8	N	N	N	N	N	N	N	38	1	>24hrs	no	N
87	Murugesan	204	69	M	Y	N	Gastric malignancy	102/74	N	92	7.5	27	0.8	N	N	N	N	N	N	N	23	5	>24hrs	no	N
88	Ganapathy	206	75	M	N	Y	gastric erosions	110/78	N	88	12.8	34	0.8	N	N	N	Y	Y	Y	Y	38	3	>24hrs	no	N
89	Thangammal	211	55	F	N	Y	gastric erosions	104/74	N	82	10.6	36	0.9	N	N	N	Y	Y	N	Y	33	2	>24hrs	no	N
90	Selvam	216	45	M	N	Y	Mallory - Weiss tear	120/82	N	78	12	26	0.7	N	N	N	N	N	N	N	36	2	>24hrs	no	N
91	Kuppuraj	217	55	M	N	Y	Esophagitis	110/72	N	72	12.4	28	0.8	N	N	N	N	N	N	N	37	3	>24hrs	no	N
92	Nanda Kumar	218	26	M	N	Y	Duodenal ulcer	108/78	N	70	13.6	32	0.8	N	N	N	N	N	N	N	39	2	>24hrs	no	N
93	Rahuman	225	42	M	Y	N	gastric erosions	120/78	N	74	12.4	34	0.9	N	N	N	N	N	N	N	37	3	>24hrs	no	N
94	Madurai Veeran	230	45	M	N	Y	Mallory - Weiss tear	116/78	N	76	12.6	28	0.7	N	N	N	N	N	N	N	38	3	>24hrs	no	N
95	Selvam	240	45	M	N	Y	Esophageal ulcer	114/80	N	80	13.6	30	0.8	N	N	N	N	N	N	N	41	2	>24hrs	no	N
96	Mohammed Ali	243	52	M	N	Y	Mallory - Weiss tear	100/70	N	82	13.6	28	0.7	N	N	N	N	N	N	N	42	3	>24hrs	no	N
97	Lakshmi	244	38	F	N	Y	Duodenal ulcer	106/76	N	88	7.5	25	0.7	N	N	N	N	N	N	N	22	2	>24hrs	no	N
98	Krishna Samy	253	60	M	N	Y	Grade lll oesophagial varices	80/60	Y	110	5.1	58	2.9	>1	2	N	N	N	N	N	16	2	<24hrs	sclero	N
99	Palanisamy	254	73	M	N	Y	Gastric malignancy	70/40	Y	104	4.5	98	4.8	>1	2	Y	N	Y	Y	N	14	6	>24hrs	no	N
100	Ram Kumar	255	27	M	N	Y	gastric erosions	116/74	N	82	13.4	34	0.8	N	N	N	N	N	N	N	39	2	>24hrs	no	N